Volume XXI. No. 8 AUGUST, 1930

Canadian Public Health Journal

Devoted to the Practice of
PREVENTIVE MEDICINE

Published by the
CANADIAN PUBLIC HEALTH ASSOCIATION
Editorial and Business Offices:
40 ELM STREET, TORONTO 2

CANADIAN PUBLIC HEALTH JOURNAL

VOL. XXI August 1930 No. 8

Why Vital Statistics?*

R. H. COATS, F.S.S., F.R.S.C.

Dominion Statistician, Ottawa
Chairman, Vital Statistics Section, Canadian Public Health Association

THOUGH my paper does not appear in black-faced type on the programme this afternoon, as a matter of fact it deals with one of the most important departures in the history of the Canadian Public Health Association—the setting up of a new and distinct section of the Association devoted to vital statistics. It is the most natural thing in the world that those responsible for such a departure, and especially the person who appears as chief figure-head in the movement, should be brought upon the carpet for an explanation. That is the real meaning of my title "Why Vital Statistics?".

In attempting to deal with so tremendous and ramifying a subject within the limits of a few minutes you will excuse me if I adopt the somewhat categorical method of the old-fashioned preacher and set out what I have to say under headings. I hasten to assure you that I will not be so long as the old-fashioned sermon; I will attempt only a limited survey.

First, then, I want to pose the question "Why vital statistics?" in a somewhat introductory and general way—from the standpoint of statistics at large.

Second, I want to say a word as to why vital statistics are collected and published by the Dominion and Provincial governments working in association in the way they do at present, and how this association came about.

Third, and last, and in the light of the preceding, I want to ask "Why vital statistics?" from the standpoint of the Canadian Public Health Association, in which we vital statisticians have now succeeded in setting up a section all our own, wherein we hope in the years to come to play a not inconspicuous or unworthy part.

In regard to my first and general approach, one is surely carrying coals to Newcastle in describing the purpose and *raison d'être* of vital statistics to the present audience. Yet because it is the well-known things of life that we often neglect and forget, I will venture at the risk of platitude to do so in a very summary manner:

*Presented at the General Sessions, 19th Annual Meeting of the Canadian Public Health Association, Toronto, May, 1930.

Vital statistics is a division of the great subject of population statistics. In almost every accounting or statistical system two kinds of records are essential: first, a day-to-day record of transactions as they occur, and second, a periodical stock-taking. In population, the stock-taking is the decennial census. The day-to-day part consists again of two records: first, the records of migration into and from the country, and second, the natural increase or decrease occurring through births and deaths, *i.e.*, Vital Statistics. The broad purpose of vital statistics is to round out and complete our purview of population as a whole, and in its fundamental aspects.

But it is the immediate and practical uses of vital statistics that concern us here and now. These immediate and practical uses are two—the legal and the scientific. The legal uses involve both the civil and the criminal side of the law. We have all of us dreamed the dream of falling heir to the millions of a long-lost uncle. But how could we keep tab on our relatives without birth certificates? How could life insurance or old age pension schemes carry on without vital statistics; they could not even draw up a list of premiums and contributions. In the field of criminal law how could society exist if all the circumstances attending so momentous a fact as death were not attested? But the great interest of vital statistics to the present audience is the scientific. Vital statistics are the measuring rod of progress for the science of Medicine and Public Health. I am repeating a well-worn story when I tell you that the death-rate in both Europe and America has been cut almost in two in the last hundred years; that the expectation of life at birth has gone up over 8 years in the United States since so recent a date as the beginning of the present century alone; that right here in Toronto infantile mortality has declined from a rate of one in every nine births only 15 years ago to one in every 14 or 15 births last year. Now it was statistics that pointed the way to this achievement. Statistics are like the captive balloon or the aeroplane that the army sends up to observe the movements of the enemy. The balloon telegraphs back its news to the artillery who straightway know where to direct their fire. All the big guns in the world are useless if you don't know where to point them. It was statistics that marked the target in the campaign against tuberculosis—now so successful. It is statistics at the present moment —statistics on a world-wide scale—that are marking the target in the attack upon the dreadful scourge of cancer. No fact of age, sex, race, or of social and economic environment, as well as of disease itself, is meaningless to preventive and curative medicine. Every time a doctor registers a death he is adding to the sum-total of the truth which alone will make us free of disease and pain.

Whilst, as I say, all this is an old, old story, I wonder if you public health officials always realize the enormous economic importance of your work—I mean the dollars and cents value of the human life which it is your supreme objective to conserve—quite apart from the humani-

tarian aspect. Man is, of course, first and foremost a living soul, but he is also a money-earner. Now, Dr. Dublin of the Metropolitan Life Insurance Company has calculated that a male human being able to earn $2,500 per year at the height of his working ability is at birth worth $9,000, at 15 worth $25,000, and at 25 worth $32,000. The female of the species may be more deadly than the male according to Rudyard Kipling but according to Dr. Dublin she is "worth" only half as much. If you evaluate the entire population on this basis some truly astonishing results are obtained. In the United States it works out that the economic value of the population as human beings is six and a half times as great as the tangible wealth of the nation in the shape of farms, mines, forests, railways, houses, goods, etc. In Canada we reckon the total value of our national wealth at about 28 billion dollars, so that even on a modest view of our economic value as individuals the total economic value of the Canadian people *qua* human beings is at least 175 billion dollars. That is the capital value of the asset it is your professional duty to maintain at its maximum of efficiency and dividend-earning capacity.

Secondly, I come to the "why" of vital statistics from the standpoint of the methods by which they are at present collected and compiled in Canada. It is an interesting and significant story. Dominion vital statistics began with a very tentative scheme set up by the Dominion Government back in the 1880's. It covered leading cities only, and it came to an end when the Provincial Registration Bureaus were organized. The latter represented an immense step forward; at the same time they implied that each province went its own way and the farther it went the more apt was it to get out of step with its fellows. Thus it came to pass that ten years ago we had nine different vital statistics acts in Canada, nine different schedules pertaining thereto, with nine different methods of administration, the resulting data being compiled in nine different ways, and published according to nine different schemes of tabulation and analysis. Thus, you could not make interprovincial comparisons—you could not even make certain inter-urban comparisons, and you could not combine the fields into a national purview. Further, you could not link up vital statistics with the statistics of immigration or with the census, the other two main divisions of population statistics as we have previously noted, because these also were carried out in water-tight compartments with methods of their own. What would you think of a business concern whose day-books did not link up with its ledgers, and neither of them with its stock sheet? We had very nearly exactly that in our population statistics, only ten years ago. But we have changed that, root and branch. In 1918 we held a conference of the registrars-general of the several provinces, the Dominion Bureau of Statistics, and various other authorities, whereat we agreed in the most excellent spirit of co-operation upon a model Vital Statistics Act; this the provinces promised to pass. Secondly, we drew up a series of standard schedules

for the registration of births and deaths, etc., which the Dominion Bureau of Statistics promised to print and supply. Finally, we agreed upon a *modus operandi* between the Dominion Bureau of Statistics and the several provincial governments whereby the latter send a copy of each and every registration to the Bureau, which compiles, tabulates, and publishes the statistics according to a scheme of presentation thoroughly threshed out between all the parties concerned, including the Dominion Department of Health. This, I can assure you, was not a light task. It took five years to put the scheme in practical operation even after we had fairly well agreed as to the lines it should follow. From 1926, however, we have had a scheme of vital statistics presented annually in a report which covers the whole Dominion, and which is on all fours with the best procedure in other countries, so that we can study our own problems in the light not only of Canadian-wide, but of world-wide experience. Thus, in this present year of grace we have achieved a real beginning in Canadian vital statistics—we have brought them to a point where for the first time they have wide significance for purposes of study and research. Canada at long last is in a position to take her place in the world movement towards the solution of some of the most important problems that confront the race of man.

This brings me to my "thirdly and lastly". For it is because of the stage now reached in our official vital statistics that there has been instituted the movement which has resulted in the addition of a Section of Vital Statistics to the Public Health Association. I ask, therefore, in conclusion, "Why vital statistics?" from the standpoint of the Association.

Well, the first answer to this question from my own standpoint is a selfish one. Vital statistics need the Association and its members. We vital statisticians can of course keep to ourselves and settle our technical problems in the cloistered seclusion of Government Departments. But while this has sufficed in the past, we realize to-day that vital statistics must establish new and practical contacts with the individuals and professions whom they serve, and that if we do not we will grow academic and sterile. Moreover, we need the day-to-day help of the medical profession. It is mainly the doctor who fills in the birth and death forms which are the *fons et origo* of all our work. On the co-operation of the doctor, therefore, we are peculiarly dependent, for though we can invoke the law, that is not the kind of co-operation that avails. We have enough self-confidence to think that if we can establish a public forum for the discussion of vital statistics we will get more of your support and appreciation for them.

The selfish reason is therefore not the real one. We are sure we offer a *quid pro quo*. If vital statistics cannot do without the medical profession, the medical profession most emphatically cannot do without vital statistics. Particularly do I believe this to be the case in that compartment of medicine dedicated to public health administration.

Here, *par excellence*, exists the need of those general measurements by which alone the success of administrative policies can be tested and tried. In the social sciences, of which public health is one, we cannot experiment as they can in chemistry, physics and the other natural sciences. We can only observe, count and analyse the doings of mankind from day to day as they adopt this, that, or the other line of policy. These observations, countings, and analyses are statistics. It was Goethe who said "Statistics govern the world", meaning simply that statistics are the only means by which we can test the validity of the principles on which we govern our conduct. It is a compelling fact that the interest of the medical profession in vital statistics, as illustrated by the number of inquiries and requests for data received at the Dominion Bureau of Statistics, has exactly trebled in the last five years.

May I conclude then on this note, namely, the service that our new Section of Vital Statistics promises to yield to the Public Health Association and to the Canadian public at this time. It is a psychological moment in the evolution of the statistics themselves. Next year we take the Census. As I said in beginning, the Census is the great stock-sheet of the Canadian population. It is the biggest of all statistical jobs—in fact, it has been called the largest single peace-time act of administration performed by the modern government; we shall need 15,000 men and women to take it, and first and last we shall spend on it nearly 3 million dollars of your money. On scores of points next year's census will provide the only social measurements we will have during the next decade of Canadian progress. The fact that we have now for the first time in Canadian history an adequate system of vital statistics to coördinate with the census, to apply to the interpretation of its results and generally to give it significance and value, is therefore a development of first importance. We should get more light and leading from the 1931 census than from any previous census in our history.

But the present is a psychological moment in an even larger and more national sense than this. Canada to-day is still one of the "new" countries—one of the "newest",—by which I mean a country of vast unoccupied spaces and undeveloped natural resources. Only half of our farm lands are "occupied", and of the latter only half are "improved". We have the second greatest timber reserve of the world and the second best fisheries. We have a quarter of a continent that is indubitably highly mineralized, the possibilities of which have only been scratched; incidentally it contains a sixth of the world's known coal reserves. Of available Canadian water-power less than a fifth has been developed. In fine, we have one-sixteenth of the earth's land surface and less than half of one per cent of the world's population. Why do I mention these facts in a paper on Vital Statistics? Simply because they mean one thing: that ultimately a great expansion of the Canadian population is inevitable—that the population problem is in

the forefront of all problems connected with the development of Canada to-day. When we emerged from the War with a debt of nearly two and a half billions compared with $336 millions in 1914, Sir Lomer Gouin declared the simple cure to be an increase of 2 or 3 millions in our population. Be that as it may, we cannot place too great emphasis on the need of studying our population and the lines its growth shall follow. I happen to be an honorary member of a committee on population set up recently by the W.S. Social Science Research Council. The first thing the Committee did was to discover and list the various researches being made by individuals or institutions in this field in the United States. How many do you think were found? No less than 384, each promising to yield some addition to the sum of knowledge regarding the human element. Is it not still more important relatively to ask *Quo Vadis* in Canada? For example, shall our increase when it comes be by immigration or by natural fertility? It is interesting to recall that Francis Walker, one of the greatest economists the United States has produced, gave it as his considered judgment many years ago that if the United States—long regarded as the greatest example in history of growth by immigration—had had no immigration whatever throughout the nineteenth century, its population nevertheless would have been as large—natural increase taking up the slack if given opportunity; and I have heard Walker's view repeated in a meeting of American scientists within the past year. That is not my own view, though I believe it to be far nearer the truth than appears on the surface. If we realized the extent to which the best of immigration is fluid (I might point out that of immigration to Canada prior to 1900 only one-sixth has proved permanently stable) we would pay more attention to vital statistics and what they have to tell us as to the prospective growth of our country.

For this great national purpose, therefore, the study of our vital statistics—the measurement of our birth-rate over our death-rate and all that this implies— morbidity no less than mortality—must be enlarged, encouraged, popularized and raised to that place in public understanding and appreciation which its importance demands. For this I can conceive no better machinery than the setting up of a public forum of discussion such as we have done to-day. Our present meeting alone has justified the resolution taken to this effect a year ago; we have had 15 papers. I am not exaggerating when I say they may represent a contribution that will compare with the sum total of previous writing of the kind in this country. That is why our present foundation of the Vital Statistics Section of the Public Health Association is a true sign of the times—one of those movements for which we can ask nothing better than that the support it receives and the success it achieves should be commensurate with its deserts. I trust the Association will take the new-born section to its heart and will foster it and nurture it till in due time it becomes an offspring of which the parent may be proud.

Poliomyelitis---A Clinical Study*

GEORGE A. CAMPBELL, B.A., M.B.,

AND

SAMUEL MIRSKY, B.Sc., M.D., C.M.,

Ottawa, Ont.

POLIOMYELITIS, being an acute systemic disease, is ushered in with symptoms of a general constitutional reaction, namely fever, headache and malaise, symptoms which are common to all acute infectious diseases and typical of none. The initial reaction may be so slight as to pass unnoticed or it may be extremely severe. Yet it is these early symptoms, which in the past obscured the diagnosis, that to-day may warn the physician of an impending catastrophe.

Other symptoms of general constitutional involvement frequently occur, but vary in different epidemics. Vomiting was a prominent prodromal symptom in the New York epidemic in 1907,[1] and in Ottawa, 1929. Diarrhoea predominated in an epidemic reported by Krause in 1909,[2] naso-pharyngitis in Muller's cases, and simple anorexia in the epidemic in Manitoba.[3]

These initial symptoms may last from a few hours to a few days, to be merged into symptoms of meningeal irritation.

Three types of cases are recognized:

1. The abortive type, which subsides without involving the nervous system.
2. The dromedary type, in which there is a return to normal for two or three days following the general systemic invasion, and then an exacerbation with symptoms indicative of meningeal involvement followed swiftly by paralysis.
3. The acute type in which the nervous system becomes involved while the general infection is at its acute stage.

The symptoms of meningeal irritation are manifest in many ways. Restlessness and irritability, hyperesthesia, drowsiness and apathy, parasthesia, spontaneous pain or pain on movement in the back or the extremities, tremors of the hands, profuse sweating and vasomotor phenomena are the symptoms common to this stage of the disease. During the epidemic in Ottawa all of these symptoms were observed and occurred with comparative frequency.[4]

Coincident with these symptoms and often before they appear, stiffness of the neck and spine occurs. The stiffness in the neck and

Read before the Joint Sessions, Canadian Public Health Association, and Ontario Health Officers' Association, Annual Meeting, Toronto, Ont., May 19th, 1930.

back is easily recognized in well-marked cases. This is considered by Draper[5] to be voluntary resistance on account of pain caused by the inflammation of the posterior spinal root ganglia. While there is rarely opisthotonos or retraction of the head, movement of the head beyond the mid-line is not permitted. More characteristic still is the "Spine Sign", best elicited with the patient sitting up in bed, his knees flexed, attempting to bend his head towards his knees. This movement usually emphasizes the stiffness of the back and is frequently associated with pain. The protective attitude of the child in sitting up, rising with his arms behind his back and then placing them in a position to prevent flexion of the spine, is significant.

Kernig's Sign as a rule is not marked. The knee jerks may be hyperactive at first and diminished later, but are not characteristic.

A cerebral tache is usually demonstrable.[6]

Often the picture in this pre-paralytic stage is characteristic. The patient appears far more seriously ill than the temperature would indicate. The face is flushed and dusky, the slight cyanosis suggesting pneumonia. There is an anxious expression associated with despair. From a drowsy apathetic state the patient is easily roused and becomes alert, but often irritable, and replies in a whining manner, preferring to be left alone. There is definite resistance of the neck and back.

The severity of the symptoms at this stage of the disease is no aid in the prognosis of the degree or extent of the paralysis, very sick cases sometimes developing no paralysis whatever, while extensive paralysis may develop with dramatic suddenness in cases with but the slightest prodromata.

The onset of paralysis is usually abrupt and often unknown to the patient until attempts at movement are made. Sometimes pain in the limb or diminished reflexes herald the onset of paralysis, particularly if muscle weakness can be detected at this stage. The detection of paresis in an unco-operative child often requires much tact and gentleness, particularly if movement of the limb is painful.

Fatal cases, which are usually due to paralysis of the diaphragm and intercostal muscles are preceded by hoarseness, dysphonia and dysphagia, the result of bulbar involvement. Yawning is a serious symptom, often a premonitor of sudden death.[7] Death may occur in six hours, but usually four or five days elapse before respirations cease.

DIAGNOSIS

The diagnosis in the pre-paralytic stage can be made clinically in well-marked cases which present the clinical picture described above. No sign or symptom, however, is pathognomonic.

Spinal fluid examination is usually helpful in the diagnosis, but renders no aid in differentiating poliomyelitis from encephalitis. The spinal fluid findings are as follows:

Appearance—Clear to faint ground glass and rarely turbid (depending on number of cells).

Cells—50 to 2,000; average 150-300. A pleocytosis of fifty per cent multilobed cells on the first day changing rapidly to a ninety per cent mononucleosis is pathognomonic. The cell count must be made at the bedside as cytolysis of the cells rapidly occurs.[8]

Globulin—considerably increased.

Pressure—elevated at first.

Sugar—Benedict's solution gives a normal reduction.

Culture—Sterile.

"The Welsbach Mantle," so characteristic of the fluid in tuberculosis meningitis is usually absent.

The cerebrospinal fluid is normal in the abortive case and during the first febrile disturbance of the dromedary type. There is usually a leucocytosis of 15,000 to 30,000.

DIFFERENTIAL DIAGNOSIS

A. Before the Onset of Paralysis—Since poliomyelitis is considered a systemic disease, early symptoms of any acute infection may simulate it. In the epidemic in Ottawa in 1929, cases of pneumococcus meningitis[9], cerebrospinal meningitis, pneumonia, pyelitis, typhoid fever, otitis media, scarlet fever, rheumatic fever were suspected of being poliomyelitis. No difficulty was encountered, however, after routine, physical and laboratory examinations were made, in differentiating these diseases from poliomyelitis.

Tuberculous and syphilitic meningitis may require differentiation. Encephalitis is often confused with poliomyelitis. Encephalitis is characterized by marked drowsiness while poliomyelitis may be associated with drowsiness but this is never so profound, and the patient is quite alert when roused. The subsequent course makes the diagnosis evident.

The bulbar type of the disease with its initial febrile disturbance followed by hoarseness, dysphonia and dysphagia may present a picture of laryngeal diphtheria, hydrophobia or tetanus.

A culture of the throat in the former and the history in the latter will aid in the diagnosis.

A large group of cases that recover without paralysis, who have low spinal fluid counts and who present all the symptoms of acute poliomyelitis, are looked upon as "abortive" cases.

Doubt often arises as to whether these cases are poliomyelitis. An absolute answer in the affirmative cannot be made. It seems quite reasonable, however, to accept them as such during the epidemic, since these cases occurred suddenly with cases that did develop paralysis and their incidence disappeared as rapidly when the epidemic subsided. If these "abortive" cases are not poliomyelitis we should have to assume

that epidemics of two diseases, poliomyelitis and some other acute disease, occurred together. Such an assumption does not seem logical.

From experiences in recent epidemics on this continent one may conclude that every febrile disturbance in children accompanied by headache, stiffness of the neck and spine, with gastro-intestinal symptoms, during the late summer months, should be suspected of being poliomyelitis. It may also be concluded that such a disturbance after the middle of November, in the temperate climate, is with equal probability not poliomyelitis.

B. After the Onset of Paralysis—Pain in the limb or diminished reflexes often precede the onset of paralysis. Muscle weakness may be detected at this stage by comparing both sides.

The detection of paresis is sometimes difficult, particularly if movement of the limb is painful. Under such circumstances one must keep in mind the possibility of other conditions which might prevent a child from moving its limb. Trauma, tuberculosis of a joint, osteomyelitis, rheumatic fever, rickets and scurvy have been mistaken for poliomyelitis.

The presence of flaccid paralysis following an acute febrile disturbance with meningismus and spinal fluid findings as described above is usually at once diagnosed as poliomyelitis. However, a flaccid paralysis may occur following diphtheria, in multiple neuritis, myelitis, obstetrical injury and hysteria. The differentiation is a neurological problem and will not be dealt with here. Facial paralysis as a result of poliomyelitis must be distinguished from Bell's palsy, or involvement of the facial nerve as a result of mumps or middle-ear disease.

TREATMENT

1. *Prophylactic*—Flexner and Stewart,[10] as the result of experiments with monkeys, have advised the use of convalescent serum in doses of 10 to 20 cc. prophylactically during an epidemic at intervals of four to six weeks.

David[11] treated contacts from every second case prophylactically with 3 cc. of convalescent serum over a five-year period. Of seventy-three treated contacts, one developed the disease. Of eighty-four untreated contacts fourteen developed the disease.

Rhoads[12] has succeeded in producing active immunity in monkeys with intradermal injections of the virus.

It is needless to add, following the work of Wickman and others, that isolation of all cases for a period of three weeks is essential.

During an epidemic all children's institutions should be quarantined to exclude visitors and operations on the nose and throat postponed.[13]

2. *Pre-paralytic*—Treatment in the pre-paralytic stage consists in administration of convalescent poliomyelitis serum.

Various methods of administration are being used.

The intrathecal use of convalescent serum as recommended by Flexner and Lewis[14] was first used by Netter[15] to treat human cases. Aycock and Luther[6] used it intrathecally and intravenously. Shaw and Thelander[16] reported a series treated intramuscularly with good results.

During the Manitoba[3] and Ottawa[4] epidemics the intramuscular route was used exclusively, and the joint experience would indicate that this method is satisfactory in the great majority of cases.

In Ottawa, 25 cc. was given intramuscularly as a routine when the supply became plentiful. Rest in bed for two weeks with careful observation of temperature, pulse and respiration, and light diet were the only other measures used. No serum sickness was noted. A few cases received a second injection.

In one case in which the child failed to respond to repeated doses of convalescent serum, rapid recovery followed blood transfusion.

3. *At the Onset of Paralysis*—If treatment is begun at the onset of paralysis, massive doses may be of some value, but no definite data are obtainable.

Postural treatment with the head well lowered is advisable prevent drowning from retained secretions in bulbar cases.[17]

One case of recovery from respiratory paralysis by means of the Drinker apparatus has been reported.[18]

4. *Post Paralytic*—Treatment in the post-paralytic stage resolves itself essentially to orthopaedic care. Absolute rest with the least possible strain on the paralysed muscles is essential. Light splints may be used. It is extremely essential to prevent deformity by proper position and support. Splints should be removed as soon as functional activity begins to return.[19]

Massage, light exercise and muscle training are very important, but these should not be begun until pain has disappeared, and at all times fatigue must be avoided.

Results with orthopaedic care reported by Dickie[20] have shown that improvement may be expected for a period of two years after the onset of paralysis.

THE STATUS OF SERUM THERAPY

Following the recognition of the preparalytic stage of poliomyelitis, treatment has been directed towards the prevention of paralysis by finding a means to arrest the disease before the spinal cord is involved.

Flexner and Amoss demonstrated the presence of hexamine in the spinal fluid of monkeys after oral administration, but its therapeutic value was found wanting in such an alkaline fluid.

Rosenow,[21, 22] claiming that a streptococcus is the cause of poliomyelitis, has made a serum and brings experimental and clinical evi-

dence as to its value. His experimental results, however, have not been corroborated by other observers, and the clinical evidence lacks convincing argument.

The use of convalescent serum is based on two conclusive experiments of Flexner and his associates[10] which demonstrate that the potent virus of poliomyelitis is rendered innocuous when mixed with convalescent serum, and that administration of the serum, as late as twenty-four hours after the monkey has been inoculated with the virus, will prevent the development of paralysis. The value of convalescent serum in human beings, however, is difficult to establish since no one can foretell in the pre-paralytic stage which case will go on to paralysis and which will not. Knowing the tragic consequences of the disease, it is impossible to treat only alternate cases for purposes of control.

Furthermore, the great variability of the severity of the disease in different epidemics and in the same epidemic renders comparison of results of treatment unsatisfactory. Ayer,[23] in a series of 126 cases, found the serum of definite value in the pre-paralytic stage. The Research Committee in Manitoba,[3] reporting on 169 cases, came to similar conclusions.

Perhaps the most convincing evidence in favour of convalescent serum is the report of Aycock and Luther[6] where 106 carefully selected cases are studied with detailed orthopedic examination. It was their conclusion that the favourable effect is shown by

1. A low mortality.
2. A low average paralysis.
3. A strikingly low paralysis of the severer grades.

In the Ottawa epidemic in 1929, Lomer and Shirreff[4] emphasized the value of treatment early in the disease by comparing the high incidence of paralysis in the first four weeks of the epidemic (before the profession and the public became aware of the early symptoms) with the low incidence of paralysis in the last nine weeks.

With the definite experimental evidence on monkeys and the favourable observations on human beings, convalescent serum is being accepted as specific therapy in the pre-paralytic stage of poliomyelitis.

The findings by Burnett and MacNamara of Australia[24], that convalescent serum prepared by the technique of the Commonwealth Serum Laboratories, without antiseptics, maintains its potency in ampoules after being stored for three years at ice-box temperature, will render the use of serum possible at all times. It should be used in every suspected case.

Experience has taught us in the epidemic of 1929 in Ottawa, that it is just as important to recognize and treat poliomyelitis in the non-paralytic stage, as it is to recognize and administer antitoxin early in diphtheria.

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The Grancher System, as Applied in the Province of Quebec, for the Protection of Childhood, against Tuberculosis*

EMILE NADEAU, M.D., D.P.H.

Assistant Director, Quebec Provincial Bureau of Health

IT has been thought that the members of our Association might be interested to hear a few remarks concerning a new venture of the Quebec Provincial Bureau of Health in connection with the prevention of tuberculosis.

It is a well-known fact that tuberculosis is still a problem in Canada and will probably remain so for some years to come. In the Province of Quebec, we have still more than 3,000 deaths *per annum* and therefore about 25,000 active cases. We may infer from this that approximately 20,000 homes or lodgings are infected with tuberculosis, in which at least from 25,000 to 30,000 children are daily exposed to contagion, owing to their lowered vitality and lack of resistance, although they are in good health at the time.

Reliable statistics have shown in France, as well as in some other countries, that out of 100 children so exposed, 60 would eventually contract the disease and 40 would die from it. These facts and figures have been carefully studied for a number of years by Professor Grancher, of Paris, a disciple of Pasteur, and he has come to the conclusion that, prevention being more economical than cure, very good results would likely be obtained if as many as possible of these contacts could be transferred from the city slums to foster homes in the country.

To carry this principle into actual practice, a voluntary association was organized, in 1903, of which Professor Grancher was president until his death in 1907. Gradually, similar associations have been created in other large cities of France and they are all helped financially by public subscriptions and special annual grants from the central government.

During the last twenty-seven years, thousands of children have been placed in country homes through these associations, and statistics, very carefully kept, have given a death-rate from tuberculosis amongst these children of less than one per cent, while 33 per cent of

*An Address presented at the Annual Meeting of the Canadian Public Health Association, Toronto, May 19th, 20th, and 21st, 1930.

them, at the age limit of 13, have decided to remain on the land instead of returning to city life.

After attending in Rome the last International Congress against Tuberculosis, Doctor Alphonse Lessard, Director of our Provincial Bureau of Health, had an opportunity to make in Paris an extensive study of the Grancher system and secure first hand information from the General Secretary of the Association, Professor Armand Delille.

Upon his return to Quebec, he submitted a full report to our Minister, the Honourable Mr. A. David, together with a proposed plan for the adaptation of the Grancher system to our local conditions. As a result, and in order to be in a position to fully justify the necessary legislation when the proper time would come, it was decided to experiment with the system upon a small scale, by placing a limited number of selected children in a few rural parishes situated in Terrebonne County, right in the Laurentian mountains, about forty-five miles from Montreal.

A public health nurse, with some years of experience in tuberculosis work, was appointed for this special work. With the co-operation of the Bruchési and Royal Edward Institutes, of Montreal, it has been possible to select from the families attending these tuberculosis dispensaries, a number of children in good health, but in daily contact with tuberculosis cases in their homes. For the time being, it was decided to take only children from 3 to 10 or 12 years of age and place a maximum of two in the same foster home, in the country. It has been the aim to elaborate a working system as simple as possible. The parents are therefore only requested to sign an agreement by which they give their consent to have one or more of their children placed in foster homes in the country, at the expense of the Provincial Bureau of Health, for an indefinite period, such agreement being subject to cancellation upon a notice of fifteen days, by either of the parties interested.

The children are then examined carefully, at the tuberculosis dispensary, any physical defects are corrected, and a medical certificate is issued on a special form to the effect that the child is in perfect health, free from any communicable disease and has been successfully vaccinated against smallpox. Steps will also be taken to have all these children vaccinated against diphtheria.

As to the selection of foster parents, it has been arranged to secure the full co-operation of the local parish priests or clergymen, who have an intimate knowledge of each of the families under their spiritual guidance. Children are placed only in highly recommended families, having no children of their own, or having grown up children, and preferably farmers. Such a list having been prepared by the parish priest or clergyman, with the co-operation of the local physician appointed to look after the children in case of illness, the selected families are visited by our public health nurse, in order to find out if the home

conditions are suitable and also to make the necessary arrangements for the care and welfare of the children. A sum of ten dollars per month, per child is paid to the foster parents for board and lodging, the necessary articles of clothing being supplied by the parents in some cases, but, as a rule, by the Bureau of Health, as well as the tuition fees, when required by the local school commission. The foster parents have also to sign an agreement by which they accept the responsibility to look after these children as if they were their own and to see that they will be properly educated, such agreement being also subject to cancellation upon a notice of fifteen days, by either of the interested parties.

The children are at all times under the supervision of our nurses who visit them as often as possible, but they receive special attention from the local priest or clergyman and the local physician, who are given a quarterly allowance as part compensation for their valuable services.

On the first Sunday of each month, the foster parents must bring these children to the church hall or the local physician's office where they are weighed and measured, the monthly cheques being distributed afterwards.

During the last three months of 1929 and the first four months of 1930, the period of our first experiment, thirty children from infected lodgings in Montreal were placed in foster homes at St. Hippolyte, Lesage and St. Jérôme, in Terrebonne County.

The scheme has worked so smoothly and the results obtained in every respect have been so encouraging for the parents, foster parents and all others interested that we had no trouble to induce our legislators to pass unanimously the necessary legislation, as Bill No. 5, sanctioned on April 4th, 1930. Articles 2 to 6 of the Act read as follows:

2. "The Lieutenant-Governor in Council may, upon the recommendation of the Provincial Secretary, establish, under the direction and control of the Provincial Bureau of Health, a service to be designated by the name of 'Child Family Placement Service'.
3. "The object of such service shall be to place in the country, with private families, children who are not tuberculous but who are threatened with becoming so through the existence of tuberculosis in their families.
4. "The Child Family Placement Service may establish, in parishes chosen by it, placement centres, and may make with the ministers of religion having the direction thereof the necessary arrangements, for the choice of the families, for the moral and physical supervision of the children who, for the purposes of the said service, shall be described under the name of pupils.
5. "The Director of the Provincial Bureau of Health is authorized to enter into the necessary contracts with the heads of the families with whom the pupils are placed for the keeping and maintenance of such pupils, to provide for payment of their board, remunerate the ministers of religion who supervise them and the physicians who may be called in to attend

them, and, in short, to provide for all expenses incurred by the said Child Family Placement Service.

6. "The sums required for operating the said Child Family Placement Service shall be taken from the consolidated revenue fund on a written demand from the Provincial Secretary, to be paid into a special fund held by the Provincial Bureau of Health for the operation and maintenance of the said service."

This last article is, I might say, very refreshing, inasmuch as we will not have constantly to worry about the necessary funds, as our friends in France always did with their voluntary association, with the result that only a limited number of children could be placed each year, on account of lack of funds. In our own case, the funds available will practically be limited only by the amount of good we will be able to do. This does not mean that it will be possible to place in foster homes all the 25,000 to 30,000 children exposed to tuberculous infection, but we may also reasonably expect that less than one per cent of the children placed will eventually die from tuberculosis, while six out of each group of ten would be likely to contract the disease and four to die from it—if left to themselves in our city slums. This, in addition to other advantages, will mean a considerable economic gain, at a very low cost, if it is considered that the system does not require any capital expenditure for special buildings, with cost of maintenance, repairs, wages, etc., which is the case for the summer camps used only for a maximum of three months each year. In addition, family life is not destroyed, but rather much improved in a good number of cases where children were under-nourished and even ill-treated in their own homes; this is to be compared too with the fact that in some instances our nurses have to kindly advise the foster parents not to spoil the children they are looking after.

As stated above, on the last day of April 1930, we had thirty children placed in four adjoining parishes of the county of Terrebonne, while a good number are on our waiting list to be placed, as soon as our organization is in good working order. Now that the necessary legislation has been enacted, we expect that before the end of the present year it will be possible to place a great number of children.

As a recruiting field, we have first the large city of Montreal where thousands of patients suffering from active tuberculosis are regularly visited by the nurses of the Bruchési and Royal Edward Institutes. This applies also to the dispensary of the Quebec Anti-tuberculosis League, for the City of Quebec. Apart from these two large cities, representing a total population of over one million people, our Provincial Bureau of Health has organized during the last few years fourteen other "T.B. dispensaries" in various centres, each with one or two full-time visiting nurses who will fully co-operate with our nurses in charge of the Child Family Placement Service. In addition, we will have on the 1st of August next, in full operation, nineteen full-time

County Health Units, for twenty-three rural counties, including a good number of fair-sized towns, a total population of 665,000 people. Travelling tuberculosis clinics are gradually being organized, generally with full-time expert clinicians, for all the municipalities included in these County Health Units, the follow-up work for the positive cases being carried out by the public health nurses of the respective Units, who will at the same time co-operate for the selection of the children exposed to contagion.

Before closing, may I be permitted to take the opportunity of our meeting at this world famous seat of learning to convey, in Doctor Lessard's as well as in my own name, the sincere thanks of the Quebec Provincial Government, for the valuable services rendered by the School of Hygiene of the University of Toronto, in giving the scientific training to our students during the last three years.

We are very proud of the fact that out of thirteen students taking the course in Public Health during the academic year just terminated, seven were young physicians from Quebec who have already been in charge or will be put in charge of County Health Units.

The science of Public Health does not know of any provincial borders and it is only through such an exchange of scientific knowledge and practical ideas that we may expect to make Canada the best country to *live young and die old*, which is the "raison d'être" of our Association.

The Health Department's Programme

"The most important task of the health department in developing a health education programme is not to take care of the unusual situation which develops during an epidemic period; it is not to describe the mechanics of operating a department or even to notify the public of the prevalence of the various communicable diseases; but rather to build up a system whereby a health consciousness will become established in the mind of each citizen."—*Established Points in a Community Programme of Health Education—Henry F. Vaughan, D.P.H.*

A Food Poisoning Epidemic* Probably Due to Cheese

R. P. HARDMAN, M.B., D.P.H. AND N. E. McKINNON, M.B.

IN June, 1929, a church social was held at X. Supper was served from 5.30 p.m. to 8 p.m., after which a concert was given. The concert was hardly more than begun when two youngsters became ill, complaining of abdominal pain. They were relieved by vomiting. In the course of the next few hours 70-75 persons, or approximately 1/3 of those present at the supper, had similar symptoms, associated in some cases with headache, diarrhoea, or prostration. The majority were fully recovered in a few hours. There were no fatalities.

In an investigation by the Provincial Department of Health, sixty-nine persons, who had been ill and who were resident in the vicinity, were interviewed personally and all details of the food eaten and symptoms suffered were obtained. Controls, 68 in number, were obtained from the same households. The controls had attended the supper but had not been sick, although they had in many cases sat beside or opposite the ones who became ill, and in many cases, too, had eaten similar articles of food from the same containers.

Table I shows the age and sex distribution of cases visited and of the controls. Of the cases, 1/3 are under 16 years of age. Of the controls approximately 1/5 are under 16. The difference in age distribution probably is not significant.

TABLE I

DISTRIBUTION OF CASES AND CONTROLS ACCORDING TO SEX AND AGE

		Under 1	1-2	3-5	6-10	11-15	16-24	25	Total	
Cases	Male		2	3	5	1	5	19	35	69
	Female			3	5	4	4	18	34	
Controls	Male	1		3	1	2	3	19	29	68
	Female			1	4	1	3	30	39	

1/3 of cases 15 years of age or under.
1/5 of controls 15 years of age or under.

The interval between the time of eating and onset of symptoms is shown in Table II. The majority, or 67 per cent, had onset of symp-

*From the Department of Epidemiology and Biometrics, School of Hygiene, University of Toronto.

toms from 3 to 5 hours after eating, a few as early as one hour, and one as late as 12 hours.

TABLE II

INTERVAL BETWEEN EATING AND ONSET OF SYMPTOMS (HOURS)

Hours	1	2	3	4	5	6	7	8	9	12
Cases	2	8	13	20	13	4	3	4	1	1
Per cent	2.9	11.6	19.	29.	19.	5.7	4.3	5.7	1.4	1.4

Abdominal pain, present in 58 cases, or 84 per cent, was colicky and in most cases was quite severe; some sought a measure of relief by rolling on the ground. Vomiting, reported in 64 cases, or 93 per cent, was said to be forceful, and in most instances gave practically immediate relief. In very few it persisted for a day or two. One case was completely prostrated, with incontinence of urine and faeces, but by next morning was practically recovered. In 42 per cent of the cases diarrhoea was reported; in most of these the onset of symptoms was prolonged. Vertigo and headache were complained of in about 40 per cent of the cases.

Ninety-four per cent were quite recovered from all symptoms by next morning. In a few who had had the most severe attacks, symptoms persisted slightly longer, or sequelae, such as muscular weakness or tingling sensations in the extremities, were noted for a few days. In one case, nephritis is said to have followed the sickness; the patient, however, was fully recovered when visited during the investigation, three weeks later.

Investigation of the Cause

The food consisted of ham, mostly in sandwiches, jellies, pickles, potato salad, cakes, pies, milk, butter, bread, cream, tea and cheese. Ice cream, candy, lemonade, and soft drinks were on sale at a canteen.

The number of cases and controls, with percentages, that used the various articles of the menu including ice cream and lemonade from the canteen are shown in Table III.

TABLE III

CASES AND CONTROLS WITH PERCENTAGES USING CERTAIN ARTICLES ON MENU

		Tea	Water	Lemonade	Bread	Cheese	Ham*	Salad	Jelly	Ice Cream	Pickles
Cases	No.	47	53	18	41	65	68	64	46	33	41
	Per cent	68	77	26	59	94	98.5	93	67	48	59
Controls	No.	49	53	16	38	35	67	59	38	34	42
	Per cent	72	78	23.5	56	51.5	97	87	56	50	62

*Mostly in sandwiches.

It is seen that three articles on the menu, potato salad, cheese, and ham were eaten by practically all the cases. Potato salad was eaten by 64 of the cases or 93 per cent, and by the controls to nearly the same extent, 59 persons or 87 per cent. As the salad came from approximately 30 different sources, no one of which would serve more than 10-20 people, and as there was no factor, ingredient, dressing, container, or server common to all the salads, this article of the menu would not seem to merit further consideration as a cause. Ham also was eaten by 68 of the cases or 98.5 per cent and by 67 controls or 97 per cent. It was, therefore, used with equal frequency by cases and controls. The ham had been bought directly from a packing house in a nearby city and was boiled for six hours on Saturday, baked in a brick oven for one hour, then set on a counter to cool for several hours during the night. At 5 a.m. Sunday, it was placed in the refrigerator from which it was removed at 3 p.m. Monday and taken directly to the gathering. There is no evidence, therefore, to suggest that ham was the factor.

In regard to cheese, however, there is a marked contrast between the percentages of cases and controls who ate it, namely 64 of the cases, or 94 per cent, compared with 35 controls, or 51.5 per cent. Cheese is, therefore, the one article of food which was used much more commonly by the cases than by those at the dinner who did not become ill. This contrast is the most striking feature of the collected data.

The details of those who gave a history of having been sick but of not having eaten cheese, are as follows:

L. P., female, age 6, had supper at 7 p.m.; ate ham sandwiches, potato salad, jelly, ice cream and drank water; complained of pain and vomiting at 10 p.m.; better in a few hours.

J. P., male, age 5, same family; had supper at 7 p.m.; ate same articles of food as L. P. except that he had no ice cream; his symptoms were similar to those of his sister with onset and recovery at the same time.

V. M., female, age 18, had supper at 8.45 p.m.; she had water, tea, ham sandwiches, two servings of potato salad, biscuits and ice cream, lemon pie and chocolate cake. She was not sick till the following morning at 9 a.m. after eating breakfast, and quickly recovered when she vomited. There was no pain.

K. M., female, age 18, had supper with V. M. at 8.45 p.m.; she had water, tea, potato salad (two servings). Her symptoms were pain and vomiting at 2 a.m. She gave a history of being very "sympathetic", and "easily upset".

It is not unreasonable to consider these four cases as merely coincidental to the epidemic—or possibly that the history of the first two is wrong as children of that age soon forget details on such occasions.

The cheese (25 pounds of a 90 pound cheese) had been bought from the local grocer, Merchant A, at X, and cut into pieces at the grounds. No *one* person had cut or handled any significant part of the cheese. Merchant A at X had bought the cheese from Merchant B at Y, who in the previous October had bought 30 September cheeses from a local cheese factory. Merchant B had retailed half of these 30 cheeses to his local customers and, with the exception of several cheeses of the lot

which were sold at a distance, had sold the remaining to various merchants in the vicinity.

Investigation soon revealed that sickness had occurred in people who had bought cheese from Merchant A at X, from Merchant B at Y and from Merchant C at Z, all within a radius of 10 miles. Through information readily given by these merchants, fifty-five such cases, where sickness followed eating the cheese, were located. The following histories are typical of those obtained from the fifty-five cases.

On May 25, cheese bought from B at Y was served at dinner at a wayside inn. Of the eleven members in the household, nine, all of whom ate cheese, were ill. Of the two who were not ill, one ate a very small piece, one did not eat any. The onset was 3-5 hours after eating, and the symptoms were similar to those experienced by those at the church supper.

From C at Z a labourer bought cheese. He and his two sons ate the cheese at noon and all were sick by 5 p.m. A third son took the cheese at 11 p.m. and was sick about 2 a.m. The rest of the family, three women, had the same food excepting the cheese and were not sick.

A farmer bought cheese from C at Z. Of eight people at dinner, seven ate cheese and all were sick within three hours. One week later the cheese was served again and was eaten by two visitors and by the "hired man", each of whom scoffed at the idea of cheese being the cause, although the "hired man" had been one of those sick the previous week. The three were sick in a few hours. A pig was given the remaining part of the cheese and was found dead 48 hours later.

Another instance is of a child of ten having two attacks following the eating, on both occasions, of cheese bought from C at Z. Severe diarrhoea followed the second attack.

Similar stories were obtained in the 55 cases, although it was readily apparent that the attack rate among those eating the cheese was by no means one hundred per cent.

This evidence, arising quite independently of, and much of it previous to the church supper, strongly supports the hypothesis that the cheese was the cause of the sickness.

Only a very crude estimate of the attack rate is possible, probably 100-200 people bought portions of the three cheeses apparently involved. Possibly 500-750 people ate the cheese. It is probable that the investigation revealed the majority but not all the cases of sickness. The attack rate, therefore, would seem to be about 25 per cent, but varied to a great extent with the piece of cheese used. Many, including Merchant B at Y, used the cheese freely and repeatedly without any ill effects.

Laboratory Investigation

Samples of the three cheeses were obtained from the merchants for investigation at two laboratories—the Provincial Department of Health laboratory and the bacteriological laboratory at the School of Hygiene and Connaught Laboratories, University of Toronto. The Provincial laboratory isolated from one sample *B. paradiffluens*. Guinea-pigs and mice inoculated with freshly isolated cultures of this micro-organism died in 24 hours but guinea-pigs inoculated with later subcultures did not succumb. In the University laboratory no micro-organism that

appeared to be significant was isolated. *B. coli*, *B. para-coli* and streptococci were found in varying proportions in different samples.

Guinea-pigs, white rats, white mice and rabbits, after previous starving for 24 hours, were fed the cheese and were given emulsions of it to drink. In some cases the emulsion was placed in the stomach with the aid of a catheter. Guinea-pigs, rabbits and mice were given, intraperitoneally, large amounts of thick emulsions of the cheese. Others were given similarly Berkefeldt filtrates of the cheese. Rabbits were given both the emulsion and Berkefeldt filtrates of the emulsion intravenously. The only casualties among these animals, numbering altogether over 50, were a few mice, from the intestinal contents and heart blood of which *B. enteriditis* was isolated. However, a few deaths occurred in control mice and from them, too, *B. enteriditis* was isolated. The mice occasionally looked sick, with fur roughened, after drinking the emulsion, but this was not sufficient evidence on which to base any definite conclusion. One rabbit that had been given the filtrate died three weeks later showing, at post mortem, pneumonia. *B. enteriditis* was isolated from the intestinal contents of this and of normal rabbits.

Eight members of the laboratory staff ate large pieces (up to 100 gms.) of the cheese—two or more times. The cheese seemed, to those eating it, to be in every respect of first class quality. Two of the eight reported nausea and vomiting following within 4-5 hours after eating very small samples of the cheese. There was no vomiting among the other six although two reported feeling somewhat indisposed with slight abdominal "rumblings" and colicky pains two to three hours after eating. This sickness however, was not at all convincing. A sample of stool from one of the members, taken 24 hours after eating the cheese, did not show any non-lactose-fermenting bacilli. The bacteriological evidence and feeding and inoculation experiments are less convincing than are the epidemiological data that cheese was the food responsible for the outbreak.

Samples of meat, bread and of potato salad were examined at the provincial laboratory, but no suggestive micro-organism was isolated from them, nor was there any evidence of any pre-formed toxin. Blood samples were obtained at the provincial laboratory from 5 cases. One serum agglutinated *B. paradiffluens* in a dilution of 1/40. When set up against *B. aertrycke*, *B. enteriditis*, *B. paratyphosus B.*, there was no evidence of agglutination.

Further Cheese Investigation

The cheese factory was visited in July; it employed one cheesemaker only and he had made the cheese in the same factory for upwards of twenty years without any previous trouble. The building, vats and equipment were scrupulously clean, as was, too, the pine floor. The rennet, colouring and culture that were used in September were from

the same bottles that were in use previous to and after September. Mice were present but to the cheesemaker's knowledge, they had never contaminated the milk or the cheese, and the possibility of such contamination seemed remote. The cheesemaker informed us that grossly dirty milk or soured milk was not accepted. Pasteurization was not practised. No processing in the making of cheese would eliminate any infection in the milk—unless it were one susceptible to the changes in acidity that take place.

Three cheeses, the number apparently involved, would be equivalent to the factory output of one day in September. This suggests the possibility that one day's lot might have been infected, but no evidence of sickness on the part of the producers or the cheesemaker in September of the previous year, could be elicited. As both readily admitted, a slight attack of diarrhoea, which did not require absence from work, might not be remembered. As all the boxes of the cheeses had been destroyed, it was impossible to check the suggestion that one day's output was responsible. The September cheese was bought by B at Y in October and stored in his cellar till the following spring. Under the circumstances, it might be just as significant that the end of the pile of cheeses in the merchant's cellar contained three which would be more exposed to the heat of the furnace than the others.

Discussion

Cheese has been reported repeatedly as being a vehicle for toxin or infection. In 100 epidemics of food poisoning in England reported by Savage and White, cheese was incriminated in eight instances. Five of these cheeses were Canadian Cheddar. In only one of these cheese epidemics was it possible, in spite of thorough investigation, to identify the causative agent. In this one instance the serum of several of the patients and of two rabbits which were inoculated with the cheese showed agglutinins for *B. suipestifer*. In extensive food poisoning epidemics attributed to cheese in Michigan in early years, 1883-1894, a toxic substance named tyrotoxicon, and later a supposedly different toxic substance, tyrotoxin, were isolated, but such substances have not been found in any instance in late years. The essential cause, therefore, of the majority of food poisoning epidemics in which cheese has been the vehicle, has not been established. Savage, who has done the most extensive work on this subject in recent years, is strongly of the opinion that the poisonous agent is a product of some of the Salmonelli group of micro-organisms. Botulism has been traced to cheese, from a sample of which *B. botulinus* was isolated. An epidemic of typhoid fever has been traced definitely to cheese. This epidemic shows the spottiness of the infection, the short incubation period, the predominance of vomiting and relative absence of diarrhoea, the absence of fatalities, the quick return to normal with few if any sequelae; these character-

istics have been noted in most of the reported cases of food poisoning attributed to cheese.

With the failure in this instance, as in most others, to demonstrate definitely an infection or a poison, either bacterial in origin or otherwise, little is accomplished by speculating as to the actual cause. The short incubation and the rapidity with which the symptoms cleared suggest, as in other epidemics, a toxin as the cause, but final decision should be postponed until more complete knowledge, epidemiological, bacteriological and experimental, is sufficient to form a sound basis for opinion.

Summary and Conclusions

An epidemic of food poisoning involving about 130 persons or more is reported. Cheese is considered to have been the vehicle for the transmission of a toxin or an infection. The exact nature of the causative factor was not determined.

The finding of *B. enteriditis*, or anti-bodies to *B. enteriditis* in test animals is without significance until the absence of such bacteria or anti-bodies previous to the test is established by trial.

With the evidence at hand, however, the question of pasteurization of the milk used for making the cheese is worthy of consideration, and investigation might be undertaken to definitely establish whether or not such a procedure would be practical.

The assistance and advice of Dr. N. H. Sutton, District Officer of Health and of Dr. Birks, Medical Officer of Health, in connection with the field investigations and of Dr. A. L. McNabb, Director, Division of Laboratories, Department of Health, Ontario and Dr. D. T. Fraser of the Connaught Laboratories in connection with the laboratory studies is gratefully acknowledged.

B. Dysenteriae Sonne Infections

MARION M. JOHNSTON, M.A. AND ALAN BROWN, M.B.

(From the Research Laboratories, Hospital for Sick Children, and the Sub-department of Pediatrics, University of Toronto)

SINCE Sonne in Denmark described the species of dysentery bacillus which bears his name, this micro-organism has been reported from Norway[1] and Scotland [2, 3, 4, 5, 6, 7] and England [8, 9, 10], and from Australia[11]. More recently Gilbert and Coleman[12] have held it to be the causal agent in cases of dysentery in New York State, although earlier workers in infantile diarrhoeas in America are believed to have isolated a species whose description is similar to that of B. dysenteriae Sonne[13]. Table I shows the number of strains isolated, the age of the patients and the type of case reported by various investigators.

TABLE I

Country	Investigator	No. of Strains	Age of Cases	Type of Case
Denmark	Sonne 1914	36	Adults and Children	Severe Mild
Norway	Thjotta 1919	25	Not given	Mild
Australia	Patterson and Williams 1922	6	Adults and Children	Severe Death
Scotland	Smith 1924	2 from 4 cases	4 cases 5 to 15 months	Mild
Scotland	Fraser et al 1926	11 strains from 33 cases	Infants and Adults	Mild
England	Channon 1926	1	Adult 30 years	Mild Long duration
Scotland	Richards 1927	4 from 5 cases	One adult Four children 7 to 13 years	Mild 3 severe
Scotland	Fyfe 1927	30 strains from 66 cases	Adults and Children	Mild
Scotland	Kerrin 1928	11	Not given	Not given Usually mild
England	Evans 1928	1	Boy 10 years	Severe Died
United States	Gilbert and Coleman 1929	12	Not given	Not given
Scotland	Hay 1930	Not given	Infants	Severe 7 deaths
Canada	Johnston and Brown 1930	20	Under 1 year to 6 years	Mild Severe 8 deaths

Twenty strains of Gram-negative "late-lactose fermenting" bacilli, which are believed to be B. dysenteriae Sonne, have been isolated in the hospital for Sick Children, Toronto. Three strains were given to us by Drs. I. H. Erb and R. B. Leacock of the Pathological Laboratory, Hospital for Sick Children, the remaining seventeen were isolated in our research laboratory. Eighteen strains were obtained in the bacteriological investigation of 175 children suffering from conditions diagnosed clinically as intestinal intoxication or infectious diarrhoea. Of these, fifteen strains were recovered from faeces, one from the faeces and the colon at autopsy, one from the gall-bladder at autopsy and one from the colon at autopsy. Two strains were isolated from pyelitis cases, one strain from faeces, the other from the culture of a catheter urine specimen. Thus a total of seventeen strains were isolated from faeces cultures.

The strains were isolated from October, 1928, to April, 1930, thirteen of which were obtained in August, September and October, 1929, while the remainder were distributed sporadically throughout the cold months of the years.

Ten patients were under one year of age, five were one year and under two, and five were in the age group, four to seven years. In this latter group were the two pyelitis cases and three mild cases of infectious diarrhoea, all of whom recovered. Eight cases of intestinal intoxication apparently due to this bacillus were very severe and all died. Six infants under one year died, also two over one but under two. It is noteworthy that the deaths occurred during the period when the greatest number of strains were isolated.

No strains of B. dysenteriae Sonne were isolated from a group of 102 control cases who belonged to the same age and social group as that which contributed our cases. These controls consisted of 9 mastoid cases, 10 pneumonia cases, 2 pyelitis cases, 17 normal infants brought to child welfare clinics, and 64 miscellaneous cases whose symptoms were not those of intestinal intoxication or infectious diarrhoea. The majority of the studies upon the control group were conducted at the season of the year when the number of strains found was greatest; the remainder were distributed throughout the years.

Methods of Isolation

Rectal swabs were taken as soon as possible after notification of the admission of a case diagnosed as intestinal intoxication or infectious diarrhoea by the medical service. The swab was incubated in nutrient broth for five or six hours prior to plating upon differential media, MacConkey's bile-salt-lactose agar, eosin-methylene-blue lactose agar, and brom-thymol-blue lactose agar being utilized. Colonies which subsequently were identified as B. dysenteriae Sonne were usually quite numerous and grew readily upon the media employed. Selected col-

onies were transferred to plain nutrient agar slopes which cultures were then used for identification. All cultures consisted of small rods, which stained uniformly and did not retain Gram's stain.

Through the kindness of Dr. David Nabarro of the Hospital for Sick Children, Great Ormond Street, London, we received six strains of B. dysenteriae Sonne. Miss M. B. Kirkbride, Division of Laboratories and Research, New York State Dept. of Health, sent us one of the strains isolated in New York State. Another strain was secured from the American type culture collection. One of the Nabarro strains, No. 681, had been isolated in 1928 from the faeces of a child who had diarrhoea accompanied by blood in the faeces. The American type culture strain No. 31 was a subculture of the strain from the Hygienic Laboratory, the original culture having come from the Lister Institute. A culture, Lister No. 2942, was received from the National Type Culture Collection, London, through Dr. R. St. John-Brooks.

Table II shows the biochemical and carbohydrate reactions of strains as described in some of the references given in this paper; it also shows our observations when the strains received from other laboratories were tested in our media, and the reactions which were given by the strains isolated in our laboratory.

For slow production of acid in media containing lactose (*i.e.* slow fermentation of lactose), the absence of motility and of gas and of indol production, the delayed acidification of litmus milk are constant characteristics of the strains as described in the literature. The strains received from other laboratories which were tested in our media conform to the description of the species in regard to these respects. Moreover it is seen that the strains isolated in this laboratory also slowly produce acid in media containing lactose, from three days onward. They are non-motile, produce neither gas nor indol, and consistently fail to cause any change in media which contains xylose, another outstanding characteristic of the species. Litmus milk was acidified slowly and, in addition, coagulated by some strains. The absence of acid production in saccharose media has been described in some reports; however, of our strains, five did slowly manifest acid production in that medium. Thjotta[1] observed that his strains formed acid in a saccharose medium. No strains blackened lead acetate although positive findings have been reported by some investigators.

Serological Tests

The Nabarro strain No. 681 was employed for the production of agglutinating serum in rabbits. The antigen was grown on an agar slope for 18 hours, washed off with sterile saline buffered with phosphate mixtures to pH 7.4; a heavy suspension was prepared and killed by heat at a temperature of 52°C. for an hour. The animal received seven injections beginning with 0.1 cc. An intravenous injection was given on each of three successive days, three days were allowed to elapse and

TABLE II

BIOCHEMICAL AND CARBOHYDRATE REACTIONS OF STRAINS DESCRIBED IN REFERENCES OF STRAINS FROM OTHER LABORATORIES, AND OF OUR STRAINS

STRAINS	Gram	Mot.	Gas	Lact.	Dex.	Malt	Sac.	Xyl	Man.	Arab.	Indol	Gel	Lead Acet.	Lit. Milk	Citrate	Jordan's tartrate
Sonne	−	−	−	+ 5-7d	+	+	−	−	+					acid		
Channon	−	−	−	+ 5-8d	+	+	−	−	+		−	−		acid		
Evans	−	−	−	+ 6d	+	+	−	−	+							
Fraser et al	−	−	−	+ 3-5d	+	+	+	−	+	+			+			
Fyfe	−	−	−	+ 3d	+	+	+ or −	−	+	+	−	−	+	acid		
Smith	−	−	−	+ 10d	+	+	+	−	+	+	−	−	+	acid		
REACTIONS OF STRAINS FROM OTHER LABORATORIES TESTED IN OUR MEDIA																
Nat. Type Cult. No. 2942	−	−	−	+ 8d	+	+	+ 13d	−	+	+	−	−	−	acid coag		
Nabarro No. 681	−	−	−	+ 7d	+	+	−	−	+	+	−	−	−	acid coag 11d	− 14d	acid
N.Y. State No. 247	−	−	−	+ 9d	+	+	+ 9d	−	+	+	−	−	very slight in 5d	acid coag 13d	− 14d	acid
Am. Type Cult. No. 31	−	−	−	+ 8d	+	+	+ 7d	−	+	+	−	−	very slight in 5d	acid coag 8d	− 14d	acid
REACTION OF STRAINS ISOLATED IN OUR RESEARCH LABORATORY																
Allan	−	−	−	+ 9d	+	+	+ 11d	−	+	+	−	−	−	acid coag		acid
Dell	−	−	−	+ 12d	+	+	− 10d	−	+	+	−	−	−	acid coag		acid
Devlin	−	−	−	+ 7d	+	+	− 9d	−	+	+	−	−	−	acid coag 13d		acid
Neill	−	−	−	+ 8d	+	+	−	−	+	+	−	−	−	acid coag 11d		acid
Freeman	−	−	−	+ 10d	+	+	− 10d	−	+	+	−	−	−	acid coag		
Holmes	−	−	−	+ 14d	+	+	+ 12d	−	+	+	−	−	−	acid 17d		
Johnston	−	−	−	+ 12d	+	+	− 10d	−	+	+	−	−	−	acid coag 15d		
Kelbie	−	−	−	+ 8d	+	+	−	−	+	+	−	−	−	acid coag		
Lithgan	−	−	−	+ 8d	+	+	+ 10d	−	+	+	−	−	−	acid coag 13d		
Lyman	−	−	−	+ 12d	+	+	− 10d	−	+	+	−	−	−	acid coag		acid
Marshall	−	−	−	+ 13d	+	+	+ 13d	−	+	+	−	−	−	acid coag 13d		acid
McCracken	−	−	−	+ 7d	+	+	−	−	+	+	−	−	−	acid coag		acid
McLaughlin	−	−	−	+ 9d	+	+	+ 12d	−	+	+	−	−	−	acid coag 18d		
McMichael	−	−	−	+ 16d	+	+	−	−	+	+	−	−	−	acid coag 10d		
Ridgers	−	−	−	+ 13d	+	+	− 10d	−	+	+	−	−	−	acid coag 10d		
Steinhoff	−	−	−	+ 9d	+	+	− 9d	−	+	+	−	−	−	acid coag 18d		
Thain	−	−	−	+ 12d	+	+	− 13d	−	+	+	−	−	−	acid 18d		
Walton	−	−	−	+ 12d	+	+	−	−	+	+	−	−	−	acid coag		
Wildridge	−	−	−	+ 10d	+	+	− 14d	−	+	+	−	−	−	11d acid coag		
Wood	−	−	−	+ 6d	+	+	− 15d	−	+	+	−	−	−	acid coag 18d		

In addition, the strains, Nabarro No. 681, N.Y. State No. 247, Am. Type Cult. No. 31, Allan, and Neill gave negative results with inosite, inulin, salicin, dulcitol and sorbitol, and positive with dextrin, galactose, levulose and rhamnose.

three more injections given, and then after another three-day period the final injection was made. The injections consisted of 0.1, 0.2, 0.2, 0.5, 0.5, 1, 1 cc. of the bacterial suspension. After a period of seven days following the final injection a trial bleed was taken and the agglutinin titre tested. The titre which reached about 1:2000 was deemed adequate, and the animal was then bled from the heart and the sterile serum stored in the ice-box. Thus immunization was accomplished within the period of approximately four weeks.

For testing the patient's cultures, the suspensions were prepared by growing fresh cultures on agar slopes, and washing the growth off with sterile buffered saline (pH 7.4). The suspensions were made up to a turbidity about equivalent to a No. 2 barium sulphate McFarland standard[14].

All strains were tested against the Nabarro anti-serum. Repeated tests yielded consistent results, that is, the strains from other laboratories and fourteen of ours (Table III) were agglutinated to full titre by this anti-serum, while six remained in uniform suspension.

TABLE III

TABLE SHOWING RESULTS OF SEROLOGICAL TESTS

Strain	Agglutination Tests			Absorption Tests	
	Serum			Nabarro No. 681 Titre after abs. by	Ridgers FII Titre after abs. by
	Nabarro No. 681	Oxford	Ridgers FII		
Nabarro No. 681	2560	320	*40	...	1600
N.Y. State No. 247	2560	160	*80	...	1600
Am. Type Cult. No. 31	2560	320	...	...	1600
Nat. Type Cult. No. 2942	2560	160	*40	...	1600
Allan	2560	320	...	...	1600
Dell	2560	320	...	...	1600
Freeman	2560	320	...	...	800
Holmes	2560	320	...	...	800
Johnston	2560	320	...	...	1600
Lithgan	2560	320	...	...	800
Marshall	2560	320	...	...	1600
McMichael	2560	320	...	...	1600
McLaughlin	2560	320	*40	...	1600
Neill	2560	320	...	...	1600
Steinhoff	2560	320	...	...	800
Thain	2560	320	...	...	1600
Wildridge	2560	320	...	...	1600
Wood	2560	160	*40	200	1600
Devlin	....	...	1280	200	....
Kelbie	....	...	1280	1600	....
Lyman	....	...	1280	1600	....
McCracken	....	...	1280	1600	....
Ridgers	....	...	1280	....	....
Walton	....	...	1280	1600	....
				Titre of Nabarro Serum =1: 2560	Titre of Ridgers Serum =1: 2000

*Incomplete agglutination in this dilution.

These six cultures constituted a group subsequently referred to as the Ridgers strains. It was found that the saline employed had to be very carefully controlled since the anti-serum gave no results with its homologous antigen, if the saline were more acid that pH 7.0. Indeed, the importance of a careful selection of sodium chloride and of pH control is shown by the fact that the Ridgers strains were clumped satisfactorily in Ridgers serum diluted with normal buffered saline but the Nabarro strains gave very indefinite results in Nabarro serum dilutions up to 1:120, and no agglutination in higher dilutions. After testing different sodium chlorides in various concentrations, and at the same time testing various pH's, a saline prepared from Merck's C.P. sodium chloride of a concentration of 0.9% and a pH of 7.4 was found quite satisfactory for both Nabarro and Ridgers strains. In the preliminary tests conducted with this saline, the control tubes which contained only saline and bacteria showed uniform suspension with no settling out of the bacterial cells. A saline of higher pH and the same sodium chloride concentration caused considerable settling of the cells. Moreover, it was considered advantageous to reduce the water-bath temperature from the ordinary 56°C. to 52°C. An incubation period of at least five hours, and usually 18 hours, was regularly allowed. When the first strains which we recovered were tested, frequently after more than five hours incubation, the racks containing the agglutination tests were removed from the water-bath to the ice-box, where they remained over night, and final readings were made on the following day. The transference of the racks to the ice-box was not found to be necessary since satisfactory results were obtained by long incubation without a period in the refrigerator. Therefore incubation was continued for 18 hours and the results were then read.

One of the strains, Ridgers FI 1, which showed no clumping in the Nabarro agglutinating serum, was employed as antigen for the production of an agglutinating serum using the same procedure as that employed for the production of the Nabarro serum.

When all the strains were tested with the Ridgers anti-serum, a striking fact was noted. The strains which were agglutinated by the Nabarro anti-serum were not clumped by this serum in any dilution between 1:40 and 1:2560. The six strains which had not been agglutinated by the Nabarro anti-serum did respond to the full titre when the Ridgers serum was used.

When an agglutinating serum which was obtained from the Standards Laboratory, Oxford, England, was used, similar results were obtained to those given by the Nabarro serum, except that the titre of this serum in our hands was somewhat lower than that of the Nabarro serum. It had apparently been produced by an antigen closely related to, probably identical with, the Nabarro No. 681 strain. The strains which fell into the Ridgers group were not agglutinated by the Oxford serum.

From these observations it appears that there are at least two definite serological groups amongst the strains isolated in this hospital, one of which includes the four strains obtained from the other laboratories. Clayton and Hunter[15] have noted that they had strains which they reported as being consistently inagglutinable. It seems, therefore, that they possibly were dealing with strains serologically different from the strain which was employed for the production of the agglutination serum with which they tested their strains.

No agglutination occurred when our strains were tested with the agglutinating sera of B. typhosus, B. paratyphosus alpha, B. paratyphosus, B. B. dysenteriae Shiga (2 strains) and B. paradysenteriae Flexner (2 strains).

Absorption tests were carried out using the formula outlined by Krumwiede[16]. The tubes containing serum and suspensions were incubated at 52°C. for at least five hours and, although there appeared to have been complete absorption, the agglutinins in the control anti-serum diluted 1:10, which was heated for the same length of time as the anti-serum which was in contact with a heavy suspension of the micro-organism under consideration, appeared to have been destroyed. The control of unheated, unabsorbed serum gave good agglutination of its homologous antigen to maximum titre. The next series of tests were carried out at ice-box temperature where contact for ten days was allowed and the mixtures of serum and suspensions were shaken each day. The titre of the anti-serum was not diminished by contact with the bacterial suspension and there was no destruction of agglutinins in diluted serum which was not in contact with any suspension. A test carried out at 37°C. showed absorption of agglutinins with no destruction of agglutinins in the control serum saline. (The special saline, having regard to the pH and the quality as outlined above, was employed for all absorption tests.) Accordingly absorption tests were subsequently incubated at this temperature for at least five hours.

The results of the agglutination and absorption tests are shown in Table III.

The absorption tests showed that both the Ridgers and Nabarro strains removed the agglutinins entirely from their homologous sera, and that the Nabarro group did not alter the titre of the Ridgers anti-serum. It was anticipated that the Ridgers strains correspondingly would not absorb the agglutinins in the Nabarro serum. Two unexpected discrepancies were noted however. Two strains, Ridgers F11 and Devlin, completely absorbed the agglutinins from the Nabarro serum thus deviating from the serological uniformity; the other four strains did not absorb the agglutinins from the Nabarro serum. In subsequent tests the Nabarro and Ridgers strains showed no evidence of absorption of agglutinins from the anti-sera of B. dysenteriae Flexner, B. typhosus Rawlings and B. coli agglutinating sera. In addition, the agglutinins of the Nabarro and Ridgers anti-sera were not absorbed by

B. dysenteriae Flexner, B. typhosus Rawlings or B. coli suspensions. It would appear, therefore, that the absorptions observed, including the absorption of the agglutinins from the Nabarro anti-serum by the Ridgers and Devlin strains, was specific.

The serum of nine patients was tested with their homologous cultures after daily subculturing in veal broth for about ten days. One serum agglutinated the suspension completely in a dilution of 1:1280, 4 in 1:160, 1 in 1:80, 1 in 1:40. Two sera were negative, possibly having been taken too early in the course of the illness; 11 sera were not obtained. If a patient's serum agglutinated the Nabarro strain, no agglutination occurred when that patient's serum was tested with the Ridgers strain. Conversely, when the patient's serum agglutinated the Ridgers strain, no agglutination occurred when that patient's serum was tested with the Nabarro strain. Correlation existed between the strain grouping and the agglutination findings when the patients' sera were tested. Thus, if a strain obtained from a patient belonged to the Nabarro group, no agglutination occurred when that patient's serum was tested with the Ridgers strain; if the strain was identified as a member of the Ridgers group, no agglutination was obtained when that patient's serum was tested with the Nabarro strain. Sera from 106 of the cases diagnosed clinically as intestinal intoxication or infectious diarrhoea were tested with the Nabarro and Ridgers strains; 29, not including the 7 noted above, showed agglutinins, 20 for the Nabarro and 9 for the Ridgers strains, in titres varying from 1:40 to 1:1280.

Other organisms such as B. dysenteriae Flexner, B. typhosus Rawlings, B. paratyphosus B., B. morgani, B. schmitzii, B. asiaticus, B. paradiffluense, B. proteus vulgaris, were not agglutinated by these patients' sera.

The sera of 7 normal infants in age from 6 weeks to 10 months were tested with the serologically different Sonne antigens with negative results. The sera of 26 control infants whose clinical condition was not diagnosed as intestinal intoxication or infectious diarrhoea gave negative results with two exceptions; one clumped the Nabarro strain in a dilution of 1:80, the other clumped the Ridgers strain at 1:40.

SUMMARY

(1) Twenty strains of B. dysenteriae Sonne have been isolated in the Hospital for Sick Children, Toronto, from 177 patients. Eighteen of these strains were obtained in the investigation of 175 cases admitted with a clinical diagnosis of either intestinal intoxication or infectious diarrhoea, two were from pyelitis cases. Faeces examinations yielded seventeen strains (one strain was isolated from faeces and colon), urine one, gall-bladder at autopsy one, colon at autopsy one. Eight strains were obtained from cases which terminated fatally.

(2) No strains were found in the examination of the faeces or autopsy material from 108 control cases.

(3) Two serological groups exist amongst strains of this species of micro-organism isolated by us.

(4) Evidence that B. dysenteriae Sonne had etiological relationship with the condition of the patients has been substantiated by the demonstration of the presence of anti-bodies in the sera of 7 cases from whom strains of B. dysenteriae Sonne were isolated. The sera of 29 additional patients from whom no strains were isolated agglutinated B. dysenteriae Sonne in dilutions from 1:40 to 1:1280. Twenty agglutinated the Nabarro strain, 9 the Ridgers strain. The absence of such anti-bodies was noted in the sera of 31 out of 33 control children.

(5) A saline which consisted of Merck's C.P. sodium chloride 0.9% and pH 7.4 was found to be most satisfactory for the serological tests. The water-bath temperature was maintained at 52°C. for the agglutination tests and at 37°C. for the absorption tests which were carried out with these strains.

ACKNOWLEDGEMENT

We express grateful appreciation to Dr. D. T. Fraser, of the School of Hygiene and Connaught Laboratories, University of Toronto, for assistance in preparation of this material and for advice and criticism during the course of this study.

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**Since going to press, another strain, which therefore is the twenty-first, has been isolated from the faeces of a 2½ year old girl whose stool contained blood. The patient's culture belonged to the Ridgers group and was agglutinated by the patient's serum in 1:640 dilution. No agglutination occurred with the Nabarro strain.*

A Report on Some Fulminating Cases of Cerebro-Spinal Fever*

WM. D. HAY, M.A., M.D.

Richardson Pathological Laboratory, Queen's University, Kingston, Ont.

FOR a period of almost two years no cases of cerebro-spinal fever had occurred in the City of Kingston or surrounding country and then suddenly during the month of June, 1928, a case† of unusual severity appeared a few miles from the city. One Saturday morning a school boy, J. S., aged 13 was going about as usual. He rode his bicycle a distance of 3 to 4 miles and afterwards took his mid-day meal. During the afternoon he complained of being tired and that his muscles were sore and stiff. He vomited during the evening. When the other members of the family went to the city he declined to go. It is interesting to note that when they returned about 10 o'clock he examined the purchases which had been made. From then on he grew rapidly worse. A physician was called and when he arrived shortly after 1 o'clock the boy was unconscious. His temperature was 104° F., pulse 160 and scarcely perceptible. Although there was no definite neck stiffness the physician suspected meningitis. He died the next morning at 5 a.m., 15 hours after the onset of illness.

The autopsy, performed while the body was still warm, showed the following appearances: A few petechial haemorrhages were present in the skin. Small petechial haemorrhages were also present in the pericardium. The lungs were intensely congested and there were extensive haemorrhages under the pleura of both lungs. Microscopically there was a marked bronchitis and Gram-negative diplococci could be seen in the exudate in the smaller bronchi. The brain was congested but otherwise normal in appearance. Smears from the base of the brain showed no pus cells. The other organs showed congestion and toxic changes. Cultures were made from the brain, spleen, nasopharynx and the heart's blood.

Meningococci were isolated from all these sources. The blood from the heart contained the organisms in large numbers. Agglutination tests were set up using these organisms and the polyvalent anti-meningococcus serum distributed by the Connaught Laboratories. The titre was low but definite as high as 1:40.

A search for carriers amongst 85 contacts of the case was made. All suspects were requested to come to the Richardson Laboratory. West's tubes were used in taking the cultures from the nasopharynx.

Presented at the Nineteenth Annual Meeting of the Canadian Public Health Association, Toronto, May, 1930.

†*Canadian Medical Journal, 1928, No. XIX, page 695, 696.*

Blood-agar plates were immediately stroked and placed in the incubator.

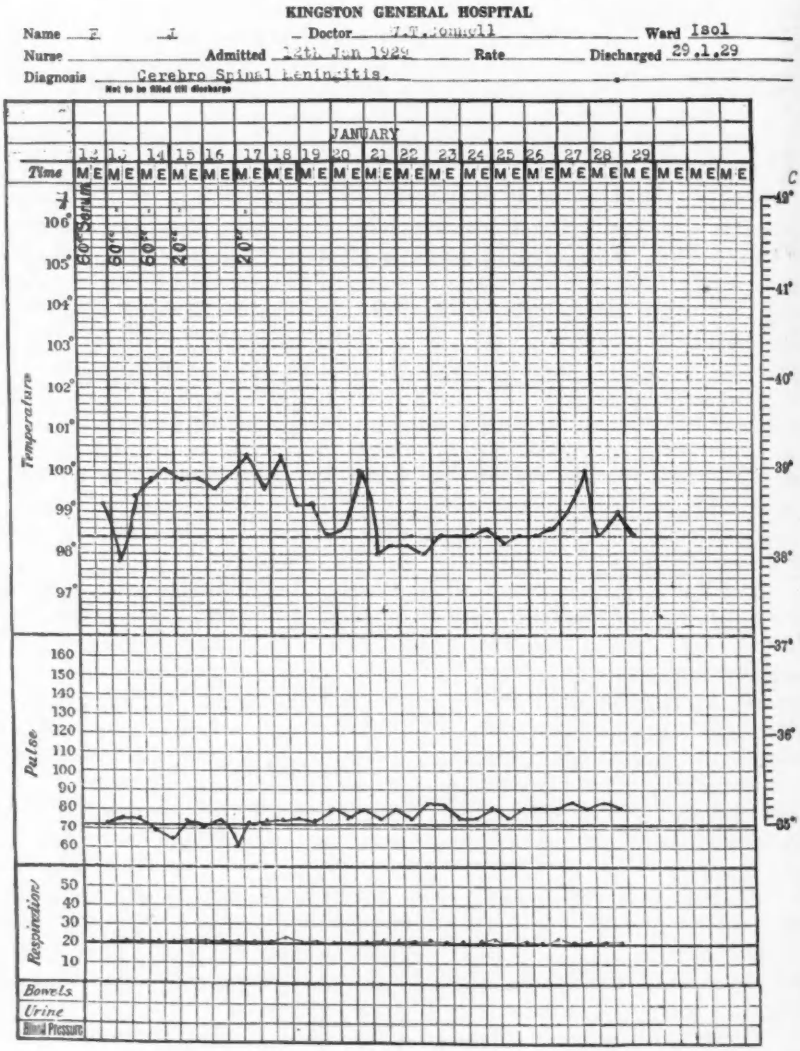

Fig. 1.—Chart of F. J., an Ordinary Case of Cerebro-Spinal Meningitis.

The plates from the father of the dead boy contained almost pure cultures of meningococci. One sister was also positive. These two carriers were taken into hospital and treated with serum. As soon as the serum rash appeared the organisms rapidly disappeared from

both and they were discharged two week after admission. No more cases occurred in the Kingston district during 1928.

On the 18th January, 1929, a school girl, F. J., aged 12 years was brought in from a neighbouring town with cerebro-spinal fever. She had been ill for three days but had not lost consciousness. During the first six days in hospital 220 cc. of anti-meningococcus serum was given. She made a complete recovery.

The hospital chart of this case illustrates the prompt response to serum therapy. This case is mentioned because it forms a good contrast with those of the fulminating type of the disease.

On the morning of the 25th January, a high-school boy, M. W., was found unconscious in his bed at his home which was about 20 miles from Kingston. The only history which could be obtained was that he had not been quite well for two days previously. Owing to the fact that his temperature was only 100° F. encephalitis lethargica was considered as a possible diagnosis. Death took place four days afterwards and at autopsy a purulent exudate was found to be widely distributed in the subarachnoid space over the brain. Smears showed typical Gram-negative diplococci. No cultures could be obtained probably because the organism died during transit to the laboratory. No carriers were found although cultures were taken from the other members of the family and also from the students of the school which the deceased had been attending.

On May 6th, 1929, a boy, E. C., aged 6, was brought to the Isolation Hospital at 5 a.m., in an unconscious condition. He was well during the previous day and had eaten very heartily. He vomited during the evening but that was attributed to overeating. He played until dark and went to bed. At 12 o'clock his sister awakened to find him delirious and throwing his arms about. The physician who was called suspected cerebro-spinal fever because it was about a year since the first case, (J. S.) mentioned above, had occurred in the next farm house, and the two families played together.

The patient was given 20 cc. of anti-serum intrathecally and 20 cc. intramuscularly within 12 hours from the onset of the illness. The doses of serum were repeated daily but the boy never regained consciousness and the numbers of meningococci in the spinal fluid continued to increase. Before death, which took place 18 days after admission, the organisms were almost as numerous as the pus cells. The patient received a total of 660 cc. of anti-meningococcus serum, half of which was given intrathecally.

This was the first opportunity which presented itself to treat one of the fulminating cases of cerebro-spinal fever with anti-serum. It was given within 12 hours from onset of illness and the dosage was considered sufficient for a child. In view of these facts Dr. W. T. Connell, Professor of Medicine, Queen's University, considered that the serum was not potent for the strain of meningococcus which was causing the fulminating cases in this district.

Another search was instituted to find carriers. A sister of the boy and the father were found to be harbouring the organisms. It is

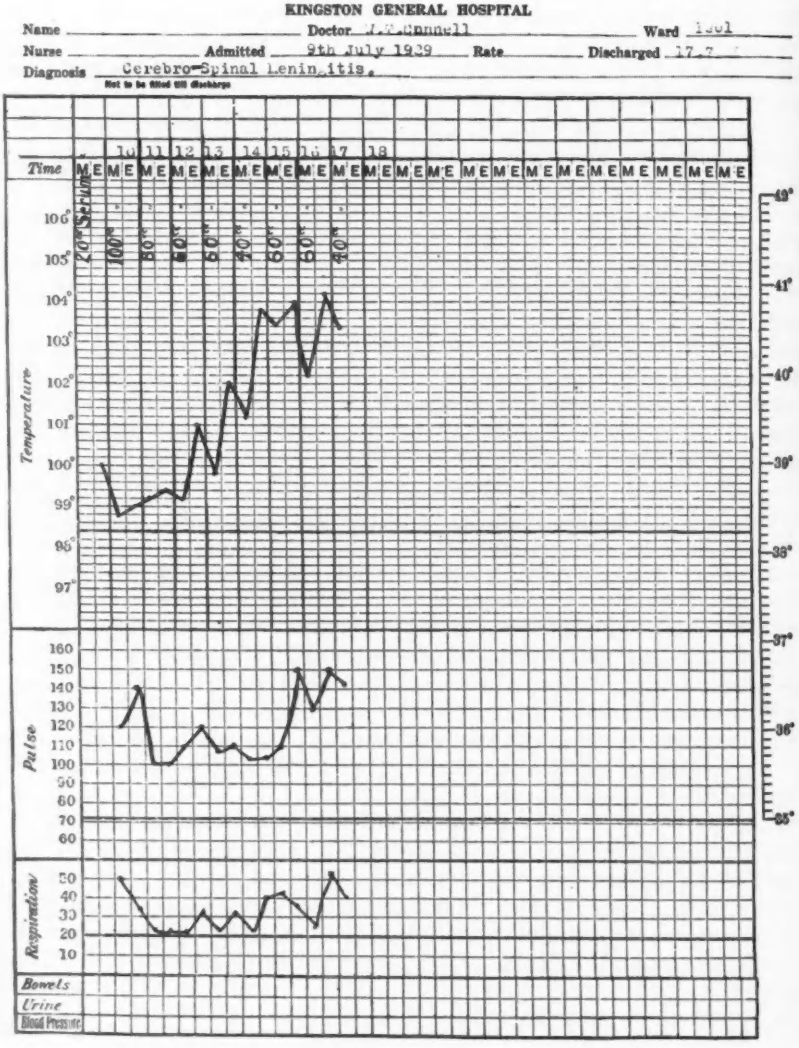

Fig. 2.—Chart of L. C., a Fulminating Case of Cerebro-Spinal Fever.

interesting to note that the two children slept together. The two carriers who were discovered almost a year previously and were treated in hospital were again examined. Much to our surprise it was found the father of the original fulminating case, J. S., was just as

active a carrier as he was when discovered the year before. However, his little girl had remained negative after leaving hospital. The health authorities requested further treatment in hospital. Another labourer who was closely associated with him was also a carrier and entered hospital for treatment. The four carriers were isolated for approximately two weeks. Negative nasopharyngeal cultures were obtained from each before they were discharged.

On July 9th, 1929, L. C. aged 18, a sister of the last case, was admitted to the hospital as a typical case of "spotted fever". She had not been quite well for two weeks and a nasopharyngeal culture had been made but no meningococci could be found. Just before admission she had become very much worse and lapsed into unconsciousness. There was rigidity of the neck and numerous petechial haemorrhages were present in the skin especially over the legs. A blood culture was carried out and meningococci were isolated. She was given 480 cc. of anti-meningococcus serum in all during the 8 days after admission to the hospital. About half of the serum was given intravenously. Some improvement took place at first and she regained consciousness. In spite of the treatment the temperature and pulse rate continued to rise and she died 9 days after admission. This was the second fulminating case to be treated without success.

The accompanying chart shows the unfavourable changes in temperature, pulse and respiration rates during her stay in hospital.

It was found that the younger sister, who was a carrier two months previously, and with whom she had been sleeping, still showed meningococci in her nasopharynx. She was again admitted to hospital for a period of two weeks. Before school opened in September the Medical Officer of Health requested that the former carriers be re-examined. None of the cultures from the suspects showed meningococci on this occasion. No new cases have occurred in that district up to date.

The cultures of meningococci from two of the fatal cases and from the associated carriers were studied by Dr. Donald Cameron of the Connaught Laboratories. The first series of agglutination tests using the polyvalent serum gave the results as shown in TableI.

TABLE I

RESULTS OF AGGLUTINATION TESTS ON THE FRESHLY ISOLATED MENINGOCOCCI

Culture No.	Anti-Meningococcus Serum Dilutions 1/50	1/100	1/200	1/400	1/500	Normal Horse Serum 1:50	Saline
54K	1	1	0	0	0	0	0
56K	1	1	0	0	0	0	0
64K	1	1	1	0	0	0	0
65K	2	3	2	1	1	0	0

4 equals complete clearing. 3 less complete, etc.

54K culture from sister of E. C. and L. C. She slept with E. C. previous to his fatal illness.

56K culture from the father of above.

64K culture from spinal fluid of E. C. a fatal case.

65K culture from carrier who harboured the organism for a year.

It will be noticed that the agglutination titre was low for all cultures. After these cultures had been repeatedly subcultured for a month they were again set up against the same serum with the following results:

TABLE II

RESULTS OF AGGLUTINATION TESTS WITH MENINGOCOCCI AFTER REPEATED SUBCULTURING

Culture No.	Anti-Meningococcus Serum Dilutions 1/50	1/100	1/200	1/500	Normal Horse Serum 1:50	Saline
54K	4	4	3	2	0	0
56K	4	4	4	3	0	0
64K	3	3	2	1	0	0
65K	3	2	1	0	0	0

It is evident that the process of subculturing for a considerable period causes the organisms to be more readily agglutinated by the anti-serum.

These four cultures were now added to stock strains used for immunizing the horses for the production of serum. Trial bleeds from the horses after a period of two months showed that the serum would agglutinate these strains of meningococci quite as well as the stock strains.

The following results were obtained:

TABLE III

AGGLUTINATION RESULTS WITH ANTI-MENINGOCOCCUS SERUM TWO MONTHS AFTER INCLUSION OF THESE STRAINS IN THE ANTIGEN

Culture No.	Polyvalent Anti-Meningococcus Serum Dilutions 1/50	1/200	1/400	1/600	1/800	Normal Horse Serum 1:50	Saline
54K	4	3	1	0	0	0	0
56K	4	4	4	4	4	1	0
64K	4	4	4	4	4	0	0
65K	4	4	3	2	1	1	0
66K	4	4	2	0	0	0	0
253K	4	4	4	2	1	3	0

66K culture from spinal fluid of L. C., a fatal case.
253K culture from blood of L. C.

This new polyvalent serum is now available but no cases of the fulminating type have appeared to serve as a clinical test of its efficiency.

An attempt was made to type four of the cultures. The following table shows the results of the agglutination tests:

RESULTS OF AGGLUTINATION TESTS OF FOUR STRAINS WITH THE FOUR MONOVALENT TYPE SERA

	TYPE I (Titre of serum 1/800)					TYPE II (Titre of serum 1/200)		
	1/50	1/100	1/300	1/500	1/800	1/50	1/100	1/200
54K	2	1	0	0	0	0	0	0
56K	0	0	0	0	0	4	4	4
64K	4	4	4	3	2	3	3	3
65K	0	0	0	0	0	0	0	0

	TYPE III (Titre of serum 1/600)				TYPE IV (Titre of serum 1/100)		
	1/50	1/100	1/300	1/600	1/50	1/100	Normal Horse Serum 1:50
54K	4	3	1	0	0	0	0
56K	3	1	0	0	0	0	0
64K	4	4	4	4	0	0	0
65K	4	3	0	0	0	0	0

Three of these cultures belong to Type III and one falls into Type II.

DISCUSSION

The above cases illustrate several important problems in the epidemiology and treatment of cerebro-spinal fever. There are four fatal cases of the fulminating type of the disease. The first one died within 15 hours from onset of illness. Two of the cases were treated with large doses of serum without success while others of less severe character were being treated with the same serum with excellent results.

Five carriers were discovered and one of these harboured the organisms for a year. The difficulties in handling carriers are great. Serum or local treatment is not always effective. The period during which persons may harbour the organisms varies greatly and hence repeated examinations at intervals of several weeks may be necessary. Carriers should not mingle intimately with other persons and should under all circumstances sleep alone. Physicians should instruct parents to have all children, who may be exposed to the disease, sleep in separate rooms.

A proper search for carriers cannot be carried on without the assistance of a laboratory. Swabs from the tonsils in our experience are useless. Satisfactory cultures can only be obtained by using West tubes. The meningococcus is a delicate organism and, therefore, the cultures must be immediately incubated. In all our investigations the contacts came to the laboratory under the direction of the local medical officer of health.

Editorials

FOOD POISONING

THE popular idea of food poisoning is frequently synonymous with that discarded expression "ptomaine poisoning". Nearly eighty years ago an Italian chemist, Selmi, introduced the term "ptomaine" ($\pi\tau\omega\mu\alpha$-corpse) to describe the basic alkaloidal products of protein putrefaction, which on injection into animals are highly toxic. This then was considered the reason for the alarming symptoms that may follow the ingestion of spoiled foods of animal origin. By a process of loose reasoning the designation "ptomaine poisoning" came to be applied to poisoning from all kinds and conditions of foods. Although ptomaines are highly toxic to animals when injected, there is little laboratory evidence to show that they are toxic when ingested. Further, since these products do not appear until the food is no longer palatable, it is highly probable that ptomaine poisoning is of very rare occurrence. This explanation of food poisoning has, therefore, fallen into discard.

With the development of bacteriology a new conception of food poisoning appeared. The relatively simple ptomaine or chemical theory was replaced by the more complex bacterial hypothesis. Earlier work incriminated micro-organisms of a few closely related species, but with the accumulation of bacteriological investigations on this subject, evidence has been produced involving an increasing variety of bacterial species, capable of growth in food products under widely different conditions. Clinically the symptoms are variable. In general, however, the cases may be classified either as "infections" or "intoxications", the former being characterized by a latent or incubation period, followed by a continued fever of greater of less duration with gastro-intestinal derangement: the latter by severe gastro-intestinal symptoms appearing shortly after the ingestion of the suspected food. Botulism, an outstanding example of food intoxication is, however, a striking exception in that gastro-intestinal symptoms are notably absent. Botulinus toxin can be prepared by growing the organism on suitable media, and its effects demonstrated in the laboratory. But can the members of the paratyphoid group, so frequently associated with food poisoning, elaborate specific toxins in contaminated food stuff?

That an outbreak may be due to a strain of micro-organisms hitherto not suspected has been recently shown by Dack, Cary, Woopert and Wiggers at the University of Chicago. A yellow haemolytic staphylococcus was isolated from the suspected food, and a toxic

substance was produced when this organism was grown in broth. Sterile filtrates caused definite symptoms of food poisoning when swallowed by volunteers. Jordan, in a further study has demonstrated that broth filtrates from various strains of staphylococci contain substances capable of producing similar symptoms in human beings.

The study of an outbreak of food poisoning in Ontario by R. P. Hardman and N. E. McKinnon, as recorded in this issue of the Journal, emphasizes the importance of careful, systematic investigation. Too often it seems that the practising physician or even the medical officer of health feels that a diagnosis of "food poisoning" and the sending of a small quantity of a suspected foodstuff to the laboratory constitutes his entire duty. It is not too much to expect that no outbreak of food poisoning should be passed by without a definite attempt being made by the attending physician and the Department of Health to ascertain the epidemiological facts. It is worth re-iterating that it is only by the close co-operation of the workers in the field and in the laboratory that our knowledge of this complex subject can be advanced.

A WORD OF WELCOME

THE Canadian Public Health Association extends cordial greetings to the members of the British Medical Association on the occasion of the holding of their Ninety-eighth Annual Meeting, this month, in the city of Winnipeg, conjointly with the Canadian Medical Association. This is the third occasion that this great body has met in Canada,—in Montreal in 1897 and in Toronto in 1906. The forthcoming session, however, is unique in the history of these two kindred associations in that it marks the first conjoint meeting since the consummation of the affiliation of our Canadian Association with the parent body.

The President of the British Medical Association, Prof. Arthur H. Burgess, of Manchester, and the President of the Canadian Medical Association, Dr. A. T. Bazin of Montreal, will have the pleasure of installing Dr. W. Harvey Smith of Winnipeg as their successor in their respective offices. Incidentally from the standpoint of Preventive Medicine, it is of special interest to note that the President-elect for 1931-32 is Dr. W. G. Willoughby, Medical Officer of Health for Eastbourne, England. Next year, then, will mark the first occasion in the ninety-eight years of this Association's development that a medical officer of health has held the distinguished office of President.

We welcome our distinguished colleagues from "home." We feel sure that the Ninety-eighth Meeting will be recorded as one of the most successful which the British Medical Association has ever convened.

LABORATORY SECTION

G. B. Reed, Ph.D., and A. L. McNabb, B.V.Sc.

THE COMPLEMENT FIXATION TEST FOR THE DIAGNOSIS OF BRUCELLIASIS*

Chas. A. Mitchell

THE discovery by Miss Alice Evans[1] that a close relationship exists between the causative organisms of *Malta fever* of humans and *infectious abortion* of cattle has stimulated a growing interest in the group to which these organisms belong. Further, the demonstration that each member of the group is pathogenic for various hosts has focused attention on brucelliasis as a public health problem.

A point of interest is the various symptoms and pathological lesions produced in the various susceptible animals. As an example, the colonization of the organisms in cattle is followed by no very definite symptoms. Thus fever is not present and practically the only clinical manifestation that suggests the presence of the organism in the host is the occurrence of abortion, this usually taking place (when it does occur) once or twice in the life of the animal. On the other hand, the colonization of the organism in humans is frequently followed by very definite and often prolonged illness accompanied by fever and prostration. Nor do the symptoms in either case lend themselves to a definite diagnosis since other bacterial agents may cause the same disturbances. It is, therefore, apparent that the diagnosis of brucelliasis is fraught with considerable difficulty and that the results of the various laboratory tests should be in the clinician's hands when the diagnosis is made. The agglutination and complement fixation tests have long been employed for the diagnosis of brucelliasis. This is more especially true with reference to the infection in domestic animals. At the present time the majority of the laboratories use only the agglutination test, since this is the simpler method, and it is believed the results are quite satisfactory.

In this Institute both the agglutination and the complement fixation tests have been employed for a number of years, and the results have been correlated, and discrepancies, where they exist, have been studied. Many sera from domesticated animals and a limited number of human sera have come under test. We discussed in a former paper[2] a few of the reasons which we believed justified the use of both serological methods. It was there pointed out that a small percentage of infected animals fail to react to one or other of the two tests, but that they usually do react to one test. In the literature in connection with human cases, attention has been directed to certain instances where the blood of individuals giving positive blood cultures failed to react to the agglutination test. It is possible if these cases had been examined by the complement fixation method a fair number would have reacted.

The following is a very brief de-

*Contribution from the Animal Diseases Research Institute, Dominion Department of Agriculture, Ottawa, Ont.

scription of the complement fixation technique for the diagnosis of brucelliasis as employed at this Institute. It is based on the method perfected by Watson[3] for the diagnosis of dourine. Those seeking details not found in this note should refer to that work.

REAGENTS USED

Haemolytic System

(1) Sheep's Red Cells:
Sheep's red blood cells washed four times and added to make a 3% solution by volume.

(2) Haemolytic Serum or Amboceptor:
Produced by the inoculation of rabbits with sheep's red blood cells. Unit value determined by titration against 5% complement.

(3) Complement:
Serum from the blood of normal guinea pigs is titred, using two units of amboceptor.

Combining System

(4) Antigen:

The preparation of antigen is one of the most important steps in the test. All strains do not produce a satisfactory antigen. In this Institute, we now use only a strain that experience has proved gives excellent results. We have not found antigen made from multiple strains more useful than that from a single strain. The

TITRATION OF DILUTED ANTIGEN

Tube	Salt Sol.	Serum Inactivated at 59° C.	Antigen	Complement		Amboceptor	3% Red Cells	
1st Set		Known Positive						
1	1 cc.	0.1	0.05	0.5		0.5	0.5	
2	1 cc.	0.1	0.1	0.5		0.5	0.5	
3	1 cc.	0.1	0.2	0.5		0.5	0.5	
4	1 cc.	0.1	0.3	0.5		0.5	0.5	
5	1 cc.	0.1	0.4	0.5		0.5	0.5	
6	1 cc.	0.1	0.5	0.5		0.5	0.5	
7	1 cc.	0.1	0.6	0.5		0.5	0.5	
8	1 cc.	0.1	0.8	0.5		0.5	0.5	
9	1 cc.	0.1	1 cc.	0.5	Incubate one hour and ten minutes	0.5	0.5	Incubate two hours
2nd Set		Known Negative						
1	1 cc.	0.1	0.05	0.5		0.5	0.5	
2	1 cc.	0.1	0.1	0.5		0.5	0.5	
3	1 cc.	0.1	0.2	0.5		0.5	0.5	
4	1 cc.	0.1	0.3	0.5		0.5	0.5	
5	1 cc.	0.1	0.4	0.5		0.5	0.5	
6	1 cc.	0.1	0.5	0.5		0.5	0.5	
7	1 cc.	0.1	0.6	0.5		0.5	0.5	
8	1 cc.	0.1	0.8	0.5		0.5	0.5	
9	1 cc.	0.1	1 cc.	0.5		0.5	0.5	
3rd Set								
1	1 cc.	—	0.05	0.5		0.5	0.5	
2	1 cc.	—	0.1	0.5		0.5	0.5	
3	1 cc.	—	0.2	0.5		0.5	0.5	
4	1 cc.	—	0.3	0.5		0.5	0.5	
5	1 cc.	—	0.4	0.5		0.5	0.5	
6	1 cc.	—	0.5	0.5		0.5	0.5	
7	1 cc.	—	0.6	0.5		0.5	0.5	
8	1 cc.	—	0.8	0.5		0.5	0.5	
9	1 cc.	—	1 cc.	0.5		0.5	0.5	
4th Set		Complement Control						
	1 cc.	—	—	0.4		0.5	0.5	
	1 cc.	—	—	0.5		0.5	0.5	
	1 cc.	—	—	0.6		0.5	0.5	

strain we use was isolated by Sir John M'Fadyean many years ago. It is free from anti-complementary substances in the ordinary dilutions, and, in addition, produces a powerful antigen. These are the two requisites.

The organisms are grown in flasks on a 3% nutrient agar slant. After three or four days the growth is washed off with a 12% saline solution to which has been added 0.5% phenol. Just sufficient saline is added to remove the growth from the media. This gives a very dense stock antigen. It is distributed in suitable vials, heated at 60 deg. C. for one hour, and stored in the refrigerator for use.

The diluted antigen used in the test is made by adding sufficient of this stock to normal saline until the opacity compares with No. 1 of the McFarlane nephelometer, and the diluted antigen is then placed in the water bath at 59 deg. C. for half an hour. It is titrated and the dose of antigen estimated. One titration per month suffices. Just sufficient is made for each day's use, a new dilution being made whenever a test is conducted.

The amount of antigen employed is that which gives complete fixation with positive serum, while three times the amount with the negative serum does not inhibit hemolysis. In no case should the amount be more than one half that which causes inhibition in the third set where serum is not present.

PREPARATION OF SERUM FOR TEST

The blood sample should be collected under aseptic conditions and the serum allowed to separate from the clot. 1 cc. or 2 cc. of clear serum is removed to a small sterile test tube and numbered for identification.

Inactivation to destroy complement and anti-complementary substances that may be present is carried out at a temperature of from 59° to 60° C., the time of exposure being one-half hour.

THE TEST

Having determined the amounts of complement and antigen to employ and prepared the serum, we are now ready to go forward with the actual test. One of the most important details in connection with the test is the amounts of serum to use. It has occasionally been found that in known infected animals, the complement fixation test failed to give a positive reaction. In these cases, paradoxically, the agglutination titre is often very high.

In our study of these apparent errors, we have found that the fault usually lies in the employment of too much serum in the test. Thus, although 0.1 cc. of serum is the favoured amount in the majority of instances—there are cases where a reaction is not given until the amount is reduced to 0.025 cc. or 0.0125 cc. Several theoretical explanations of this phenomena have been given, but it is not necessary to consider them here. In the test, three dilutions of serum should, therefore, be employed, 0.1 cc., 0.025 cc., and 0.0125 cc., with a suitable control for each. Known positive and negative sera should always be included as controls.

Assuming that the complement and antigen have been titred, and the amounts to use determined, and that the serum has been inactivated, the following table will give in a condensed form the method of procedure for a diagnostic test:

Tube No.	Salt Sol.	Serum (inactivated at 59° C.)	Complement	Antigen		Amboceptor	3% Red Cells	
		Serum under Study						
1.	1 cc.	0.1 cc.	0.5	0.3		0.5	0.5	
2.	1 cc.	0.025 "	0.5	0.3		0.5	0.5	
3.	1 cc.	0.0125 "	0.5	0.3		0.5	0.5	
4.	1 cc.	0.1 "	0.5			0.5	0.5	
5.	1 cc.	0.025 "	0.5			0.5	0.5	
6.	1 cc.	0.0125 "	0.5			0.5	0.5	
		Known Positive Serum						
1.	1 cc.	0.1 cc.	0.5	0.3		0.5	0.5	
2.	1 cc.	0.025 "	0.5	0.3		0.5	0.5	
3.	1 cc.	0.0125 "	0.5	0.3		0.5	0.5	
4.	1 cc.	0.1 "	0.5			0.5	0.5	
5.	1 cc.	0.025 "	0.5			0.5	0.5	
6.	1 cc.	0.0125 "	0.5			0.5	0.5	
		Known Negative Serum						
1.	1 cc.	0.1 cc.	0.5	0.3		0.5	0.5	
2.	1 cc.	0.025 "	0.5	0.3		0.5	0.5	
3.	1 cc.	0.0125 "	0.5	0.3		0.5	0.5	
4.	1 cc.	0.1 "	0.5			0.5	0.5	
5.	1 cc.	0.025 "	0.5			0.5	0.5	
6.	1 cc.	0.0125 "	0.5			0.5	0.5	

Incubate one hour and ten minutes

Incubate two hours

After removing the tubes from the incubator, the results of the test are recorded. The tubes are then placed in the refrigerator over night. In the morning, the cells have settled, and the different degrees of reaction are more apparent.

The degrees of reaction are recorded in the following way:

\- *Negative reaction,* complete haemolysis.

\+ *Faint positive reaction*— almost complete haemolysis, but trace of cells at the bottom of the tube.

++ *Weak positive*—about one half of cells haemolysed.

+++ *Well marked positive* — nearly all red cells at bottom of tube, just a tinge of haemolysis in the supernatant fluid.

++++ *Strong positive reaction*— red cells in mass at bottom, supernatant fluid water clear.

INTERPRETATION OF REACTION

The first care of the serologist should be to examine the serum control tubes, that is, the tubes in his test which contain no antigen. It is imperative that in these tubes, complete haemolysis should take place, since this is evidence that the haemolytic system is sufficiently strong, and that the serum itself contains no thermostabile anti-complementary substances. If reactions take place in these tubes, the test is worthless and should be repeated, probably with new serum, at a later date. In very few instances is this necessary.

When the serum controls indicate that no non-specific reaction has taken place, the next step is to consider the degrees of reaction and the dilutions in which these take place. Generally speaking, little difficulty is encountered in this by the trained serologist, but for those with little experience, it is often difficult to interpret the results.

When strong reactions (++++) are found in all dilutions, and complete haemolysis is present in the serum controls, or when a strong reaction (++++) or a well-marked positive reaction (+++) is found in the first tube, and in decreasing amounts, or absent in the two second dilutions, this should be considered as positive evidence of infection. A weak-positive reaction (++), or a faint reaction (+), in any one of the three tubes, should be looked upon as a questionable reaction, and as suspicious of infection. A new sample of serum should be obtained in a few days, and again examined.

The serologist should also keep in mind the phenomenon to which reference has already been made, and which, in a small percentage of cases, is found,—that is, in the first dilution complete haemolysis may take place, while in the two succeeding dilutions (or even in the 0.0125 dilution alone) reaction takes place. It is our custom in cases of this kind to make a second test, using eleven dilutions, commencing at 0.1 cc., and in each succeeding dilution, using just half the serum of the former. In this manner the reaction is brought within the range of certain dilutions, and when it takes place one should have no hesitation in considering it as evidence of infection.

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REPORTED CASES OF CERTAIN COMMUNICABLE DISEASES IN CANADA* BY PROVINCES—JUNE, 1930

Disease	P.E.I.	Nova Scotia	New Bruns	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia
Diphtheria	1	20	6	127	237	87	16	21	23
Scarlet Fever	6	65	15	251	511	30	61	45	49
Measles	—	13	—	335	1,319	195	185	14	73
Whooping Cough	—	6	—	89	232	113	66	7	217
German Measles	—	2	—	119	429	†	31	1	6
Mumps	—	3	—	187	130	201	44	9	48
Smallpox	—	—	—	6	47	4	22	—	1
Cerebrospinal Meningitis	—	2	—	4	11	—	1	1	1
Anterior Poliomyelitis	1	—	—	3	2	—	—	2	—
Typhoid Fever	—	1	8	36	30	1	7	—	6

*Data furnished by the Dominion Bureau of Statistics, Ottawa.
†Not reportable.

NEWS AND COMMENTS

P. A. T. Sneath, M.D., D.P.H.

Beauce County Health Unit Receives a Visit from His Excellency the Governor-General

A most gratifying recognition of the importance of public health work in general and its particular application in the County Health Unit Scheme was accorded by the visit on July 17th last, of His Excellency the Governor-General and the Viscountess Willingdon, to the Beauce County Health Unit, accompanied by Dr. Alphonse Lessard, Director of the Provincial Health Service, and Mme. Lessard. His Excellency and party motored from Quebec to Beauceville, arriving about 3 p.m., and were received by M. H. R. Renaud, the Mayor, who read an address of welcome, to which reply was made in French, after which Dr. Deschenes, the Director of the Beauce County Unit and his staff were presented. A detailed inspection of the headquarters of the Unit was made, special interest being shown in the portable X-ray equipment under the charge of Dr. Guerard. Their Excellencies' attention was particularly directed to the work in Schools, Babies' Clinics, Tuberculosis Clinics and Diphtheria immunization, incidental to the latter of which, the small daughter of M. Ed. Fortin, M.P., was given her first injection by way of demonstration. Interest was expressed on being informed that during the month of June, the nursing personnel had made 2,235 visits to the homes in the Unit, and that the diphtheria immunization campaign instituted three weeks before had already reached nearly 1,500 children.

The Beauce County Health Unit was the first of its kind in Canada. So successful has been this initial demonstration of the "life-and-health-conservation" value by this Unit that the Province of Quebec, with the munificent assistance of the Rockefeller Foundation and the various municipalities, has since established eighteen other full-time health units, notice of which has been made from time to time in these columns. Further, other provinces, British Columbia, Saskatchewan and Manitoba, have followed the lead of Quebec, although none of the others have as yet established so many units.

For years it has been felt that the ultimate solution of the rural health problems of Canada lies in the institution of full-time public health work, along lines similar to that afforded the urban areas. Public sentiment in Canada, stimulated doubtless, by the successful demonstrations of the solution to these problems in Quebec and elsewhere, is such, that the subject was raised before the Dominion House of Commons by Henry Spencer, M.P. (U.F.A., Battle River) and, prior to the prorogation of the House in July, Parliament was considering the advisability of making appropriations to assist in the establishment of County Health Units throughout the Dominion.

The gratification and interest of His Excellency the Governor-General and Lady Willingdon in the thoroughness and comprehensiveness of the work being done in Beauce County adds further influence to the inclina-

tions of the citizens of the Dominion in their desire for the betterment of the public health in rural communities.

Proposals for a General Medical Service in Great Britain

IT may be remembered that prior to Mr. Lloyd George's introduction of the National Health Insurance Scheme in 1912, the organized medical profession of Great Britain, through the agency of the British Medical Association, was not prepared to advise government in respect of the medical aspects of such a tremendous though admittedly restricted experiment in the socialization of medical services, and as a result has been obliged ever since to seek various modifications to the Act to increase its efficiency. The British Medical Association is now, however, presenting carefully studied "Proposals for a General Medical Service for the Nation", in view of the gradual drift of public opinion towards the extension of medical services to a larger group of the population than is now served by the National Health Insurance Act. These proposals are presented to the Association as an appendix to the Annual Report of Council for 1929-30, as a framework for the purpose of eliciting criticism from the public upon which more adequate medical supervision and treatment may be afforded to the greatest numbers within the nation.

The fundamental principles upon which this report is based are probably sufficient to meet the needs of this section, and in outline show the trend of thought in these "Proposals." They are as follows:—

I. "That a satisfactory system of medical service must be directed to the prevention of disease no less than to the relief of individual sufferers.

II. "That the medical service of the community must be based on the provision for every individual of a general practitioner or family doctor.

III. "That a consultant service and all necessary specialist and auxiliary forms of diagnosis and treatment should be available for the individual patient, normally through the agency of the family doctor.

IV. "That the interposition of any third party between the doctor and patient, so far as actual medical attendance is concerned, shall be as limited as possible.

V. "That, as regards the control of the purely professional side of the service, the guaranteeing of the quality of service, and the discipline of the doctors taking part in it, as much responsibility as possible should be placed on the organized medical profession.

VI. "That in any arrangements made for communal or subsidized or insurance medical service, the organized medical profession should be freely consulted from the outset on all professional matters by those responsible for the financial and administrative control of that service.

VII. "That medical benefits of the present National Health Insurance Acts should be extended so as to include the dependents of all persons insured thereunder.

VIII. "That every effort should be made to provide medical and nursing service facilities in institutions (Home Hospitals) where the family doctor may treat those of his own patients who need such provision and who can thus remain under his care."

Prince Edward Island

A joint dinner of the Gyro and Rotary Clubs was held at the Beach Grove Inn at Charlottetown on the evening of July 7th, at which there were five speakers on public health problems. These were, Dr. R. E. Wodehouse, Secretary of the Canadian Tuberculosis Association, Dr. A. T. Bazin, president of the Canadian Medical Association, Messrs. Parker and Smith of the Life Officers' Association of Toronto and the Hon. Mr. Lea, Premier of the Province.

The Annual Meeting of the Prince Edward Island Medical Association was held recently. Dr. P. McIntyre of Montague and Dr. A. T. Bazin of Montreal spoke on Periodic Health Examination. In the afternoon the following papers were presented:— "Common Diseases of Children", by Dr. F. W. Tidmarsh, Charlottetown; "Peritonitis with Post Mortem Findings", by Dr. R. F. Seaman of Charlottetown; "Surgical Treatment of Diabetes", by Dr. A. T. Bazin of Montreal, and "Tuberculosis", by Dr. R. E. Wodehouse of Ottawa Ont.

The following officers were elected for the ensuing year:— President, Dr. J. E. Fleming; Vice-Presidents, Queen's County, Dr. J. P. Lantz, Charlottetown; Prince County, Dr. J. C. Simpson, Summerside; and King's County, Dr. Lester Brehaut, Murray River; Secretary, Dr. J. W. MacKenzie.

Ontario

AT the Annual Meeting of the Social Hygiene Council in Toronto, the following officers were elected:— President, W. H. Shaw, Esq.; Vice-Presidents, Dr. A. J. MacKenzie, and W. A. Peace, Esq.; Honorary Treasurer, E. J. Howson, Esq., and Secretary, Dr. C. P. Fenwick.

Arrangements are being completed for the formal opening of the Banting Institute, in the University of Toronto, by Lord Moynihan of Leeds, England, on September 16th, 1930. The Banting Institute is situated on College Street, opposite the Toronto General Hospital, with which it is connected by tunnel. The building, of modern Georgian style, provides ample facilities for research and teaching purposes, giving accommodation for the work being carried out under the Banting and Best Chair of Medical Research, in addition to the Departments of Medicine, Surgery, Obstetrics and Gynaecology, Pathology and Bacteriology, and Pathological Chemistry.

In conjunction with this event a special Convocation of the University of Toronto will be held, at which several honorary degrees will be conferred.

Manitoba

THE Manitoba Hospitals' Association met in Brandon on July 10th last. The morning session was devoted to the presentation of reports, during the afternoon the session was occupied by a round table discussion on hospital problems. The evening session was arranged as a dinner, followed by a social gathering.

Saskatchewan

DR. C. L. HAMES has been appointed Director of Child Welfare with the Department of Public Health.

BOOK REVIEWS

D. T. Fraser, B.A., M.B., D.P.H.; R. R. McClenahan, B.A., M.B., D.P.H.

Tuberculosis Among Children—*By J. Arthur Myers, Ph.D., M.D., F.A.C.P., Chief of Medical Staff, Lymanhurst School for Tuberculous Children, Minneapolis. Publishers, Charles C. Thomas, Springfield, Illinois, and Baltimore, Maryland.* 208 *pages. Price* $3.50.

This book is conveniently divided into three parts. Part I deals with Tuberculosis in Infancy with a chapter on Tuberculous Meningitis by C. A. Stewart, M.D., Ph.D. Part II deals with Tuberculosis in Childhood with chapters on Bone and Joint Lesions, by Paul W. Giessler, M.D., and on Chronic Non-Tuberculous Basal Pulmonary Diseases in Childhood, by C. A. Stewart. Part III discusses tuberculosis in the teen ages. The book thus embraces the entire scope of this disease in its different phases as seen in children. At the conclusion of each chapter the text is well summarized, and there is an abundance of references.

The introduction by Allen K. Krause, M.D., sounds the hopeful note concerning this disease in children, and may be summed up in his own words: "Such a set of facts must mean nothing less than exceptionally good tolerance of tuberculosis by the child."

The author points out that the common belief among physicians that tuberculous infection is universal in later childhood is one of the great handicaps in combating tuberculosis to-day. Recent surveys have shown the incidence in some areas to be as low as 10 per cent.—hence the increased value of the tuberculin test. In respect of this test the author gives a great deal of detail as to both the technique of performing it and the interpretation. The colour plate of the Mantoux method is particularly good.

In a paragraph, "Determination of Type of Bacilli not Always Possible"—it is stated that "the type of tubercle bacilli causing the infection cannot be determined with a high degree of accuracy by the tuberculin test." We believe it is generally accepted that it is not at all possible to determine, by the tuberculin test, the type of tubercle bacillus causing the infection.

Childhood Tuberculosis is definitely defined in accordance with the resolution adopted by the American Sanatorium Association in May, 1929, *i.e.*, that "Childhood Type Tuberculosis" be used to describe the diffuse or focal lesions in the lungs and adjacent tracheo-bronchial nodes that result from a first infection of the pulmonary tissue with the tubercle bacilli." This immediately removes all confusion as to terms and makes the reading of this book particularly delightful.

The much debated point that the adult type of pulmonary tuberculosis results from the childhood type is dealt with in the following: "more recent evidence, however, leads us to believe that the adult type of tuberculosis so often seen in the teen ages is frequently due to an exogeneous source of infection. In fact, the adult

type of lesion quite commonly develops in the lung opposite to that containing the childhood type." The danger to children from tuberculosis in elderly people is aptly described and illustrated. Reference is again made to the tuberculin test—in that it is of much value among the aged; some have not been infected, in others it may have "burned out". Thus a negative test is of great importance.

In all, this is an exceedingly satisfactory volume, filled with pertinent facts of the various phases of this great problem; a book heartily recommended to all those interested in tuberculosis.

M. H. BROWN

The Bacteriophage and Its Clinical Applications—*By F. d'Herelle, Professor of Bacteriology, Yale University School of Medicine, translated by George H. Smith, Professor of Immunology, Yale University School of Medicine. Published by Charles C. Thomas, Springfield, Illinois and Baltimore, Maryland.* 254 *pages. Price,* $4.00.

In the preface, having first disposed of the physicists and chemists of the nineteenth century, Dr. d'Herelle says: "As the last century closed—that period of blissful satisfaction—the biologists also had erected a splendid structure into the foundations of which they had harmoniously interlocked the cellular theory of life, the theory of the fixity of bacterial species and that of the "antibodies" ornamented with "side-chains" such as would explain recovery and all immunity. Suddenly bacteriophagy made its appearance. The structure could not support the added weight of the new facts: it crumbled. The cellular theory of life is manifestly false, for life is an attribute of infracellular particles. The antibodies play no part in the phenomena of recovery. The form and the properties of bacteria are inherently variable characters.

"In the pages which follow an attempt has been made to explain as simply as possible the extremely complicated subject of bacteriophagy. An effort has been made to make the text understandable to all intelligent persons, although it is addressed especially to practitioners of medicine."

In the text which follows, the author treats each phase of the subject considered in as definite—and as entertaining—a manner. A short review of this character does not permit a statement of the various views advanced, much less any argument or attempt at refutation. The orthodox bacteriologist and more especially the immunologist, will find material for argument—and for thought, too—on every page. New material, much of it as controversial as the old, has been included. To get d'Herelle's views of the bacteriophage, it is necessary to read the book. And the reading of it will be found stimulating, perhaps at times irritating.

The pleasing set-up and printing and the absence of typographical errors are worthy of special mention.

This book should be widely read by those with critical but open minds.

P. A. T. SNEATH

CURRENT HEALTH LITERATURE

D. T. Fraser, B.A., M.B., D.P.H.

An Analysis of the Influence of Irradiation by Means of a Mercury Vapour Lamp upon the Health and Fertility of a Breeding Stock of Guinea-pigs and upon the Health of their Offspring during the First Six Weeks of Life.—*Petrie, G. F., J. Hyg., XXX*: 2, *June*, 1930, *pp.* 154-163.

The guinea-pigs at the Lister Institute were suffering from a pneumococcal infection, mainly incident upon the breeding sows. The mortality showed an increase in the first quarter of the year, this seasonal selection suggesting a depletion of vitamin reserve, and vitamin D was considered worthy of investigation.

The investigation involved 4,233 animals, 613 adults and 3,620 young during the first six weeks of life, and extended from February, 1928, to February, 1929, with an added period of three months for observation after cessation of irradiation.

Irradiation was carried out by means of a mercury vapour quartz burner and varied from 1 minute per day to 5 minutes four times a week, 2 minutes per day twice a week, and 3 minutes per day for 6 days per week.

The conclusion is as follows: The experiment furnishes no evidence that irradiation of a breeding stock of guinea-pigs with ultraviolet light from a mercury vapour lamp exerts any favourable influence upon (1), the general mortality, the susceptibility to a spontaneous pneumococcal infection, and the fertility of the adult animals; and (2), the survival-rate of the young in the early weeks of life, their nutrition *in utero* as indicated by the weight at birth, and their susceptibility to the pneumococcal infection.

On the Effect of Ultra-Violet Irradiation upon the Resistance of Mice Exposed to Pasteurella Infection. — *Hill, Greenwood and Topley. Br. Jour. Exp. Path., Vol. XI, No.* 3, *June*, 1930.

In the author's words: "The object of the experiment described in this paper was to test upon a numerically adequate sample of animals exposed to infection under approximately normal conditions whether irradiation conferred any immunity from fatal infection."

An infected herd was formed by inoculating 25 mice with *Pasteurella* and adding them to 100 normal mice. Normal mice were then added daily to the infected herd. When infection spread to the normals, irradiated mice and normal mice were added daily to the herd. Deaths were recorded and life tables for both groups prepared. The experiment involved more than 1,000 mice, and extended over a period of seven months. The conclusion is that the controls lived rather longer than the irradiated mice when all causes of death are brought into the account, and quite as long when only the specific cause of mortality is considered. It is quite clear that irradiation conferred no advantage upon the treated animals in respect of the hazards to which they were exposed.